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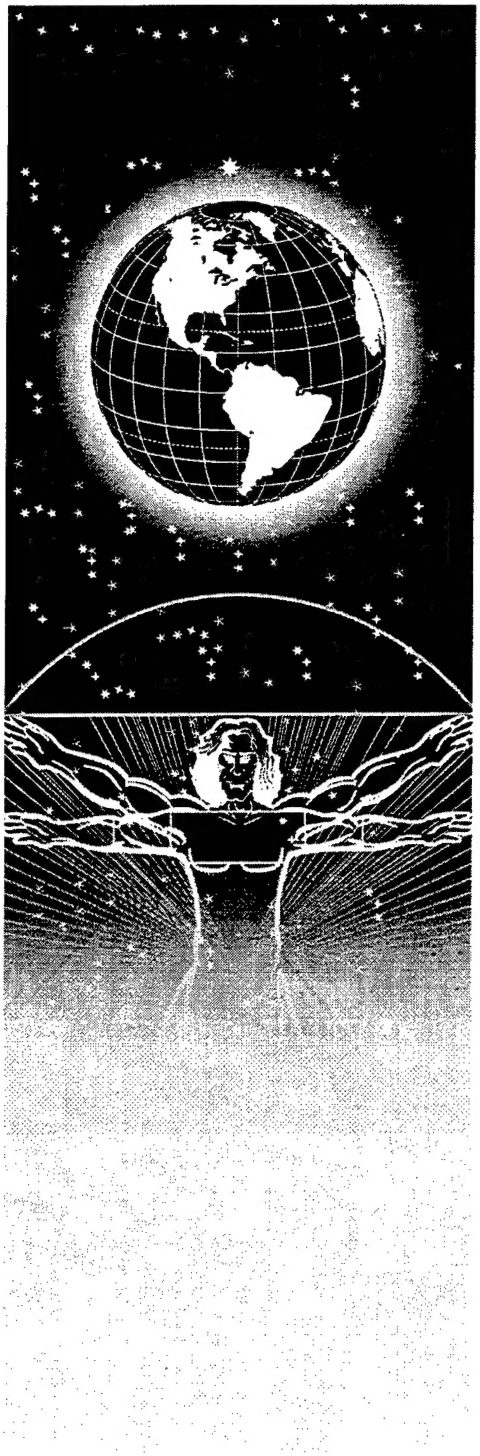

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Human Pulmonary Tolerance to Dynamic Over-Pressure

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November 1996

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This report has been reviewed and is approved for publication.

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The literature was reviewed for animal and human data defining the limits of dynamic pulmonary overpressure. The physiology and basic theory of decompression are discussed. The current understanding of static pressure limits is discussed, and its basis in the literature identified. The maximum pressure that can be safely tolerated by the human pulmonary system in a dynamic over-pressure situation is unknown. This measurement, in general, has not been performed. Evidence is presented which suggests that the unsupported chest wall of the human population can safely support 80 mmHg static and dynamic over-pressure of the lungs. Safe static pressure in the human population wearing chest and abdomen support devices is at least 190 mmHg. It is probable that a pilot without pulmonary pathology, wearing well designed life support equipment, could support higher pressures without permanent injury. The problem lies in the lack of human data, partly due to the fact that humans have never expected to be exposed to decompression in excess of the data presented in this summary.

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"It is now generally realized that before going any further with design and construction, the industry must ascertain from the physiologist whether the operating conditions planned are favorable for the comfort and well being of the human organism." --J.W. Heim January 27, 1938[72]

INTRODUCTION

Manned flight in the new generation of fighter and attack aircraft will be physiologically more demanding than any other aircraft in the inventory.[5,50] Restrictions placed upon life support equipment in order to achieve performance objectives place a premium upon all equipment used, and has forced a re-examination of currently imposed safety limits.[1,120] With increased operational capability, including regular flights to over 50,000 feet projected, pressure suits, and/or increased cockpit differential pressures must be used to support the pilot.[8,24,43,57,76,77] Pulmonary overpressures from decompression events will occur.[83,85,86] In addition, positive pressure breathing for G protection is being further developed, refined, and employed, which also places pilots at risk for overpressure incidents.[14,35,80,107,108] Static pulmonary pressure limitations are being applied to situations where dynamic pressure limitations are more appropriate. The purpose of this study is to determine exactly what dynamic over-pressures are compatible with life. The limit of dynamic pulmonary over-pressurization is to be identified ideally with reference to the population of interest, the current United States military aviator, and is not meant to be applied to the general population in a clinical medicine setting. Ideally, this dynamic pressure limitation should be isolated from the physiologic confounders of high sustained G, hypoxia, and the low pressure effects of decompression sickness and ebullism.

Normal Pulmonary Mechanics

Normal pulmonary mechanics are explored in order to refresh the reader with concepts important to this research. Pulmonary mechanics includes the study of the forces which move the lung and chest wall, the resistances they overcome, and the resulting flows. [119] It is useful to briefly review some of the basics of the elastic properties of the lung and chest wall, and airway resistance.[22]

The lungs are bounded visceral and parietal pleura which are supported by the ribs and their intercostal muscles, spine, sternum, diaphragm and mediastinum. The diaphragm and the intercostal muscles are the primary muscles of respiration. There are accessory muscles which are used during periods of active respiration, such as during exercise and forced expiration. These accessory muscles are recruited during positive pressure breathing. Abdominal contents can also provide support to the diaphragm, forcing it up and limiting its excursion. This is accomplished through the rectus abdominous muscle group physiologically, and with abdominal bladders in the anti-G suit.

During normal, quiet breathing, inspiration is active, and expiration is passive. During inspiration, the diaphragm and external intercostal muscles contract, enlarging the internal volume of the thorax, and in turn the lungs, creating a slight negative pressure. Air moves across a pressure gradient, from high to low pressure, and moves into the lungs. During exhalation, the muscles relax, creating a slight positive pressure, moving air out of the lungs and allowing the thorax to return to its neutral state. The pressures in this cycle are compared to the ambient atmospheric pressure. A positive pressure is a pressure greater than ambient, and a negative pressure is a pressure less than ambient.

The elastic properties of the respiratory system are a composite of the elastic properties of the lungs and the thorax. The forces generated by each component's elastic tendencies vary with the volume in the system, and are non-linear.[63,84,103,118] The volume change per unit of pressure change is referred to as the *compliance* of the lung. At full volume, the total lung capacity(TLC), the compliance is reduced, as further increases in pressure can only increase volume through damage--air going outside of the pulmonary system. Compliance is normally measured at the point where the elastic tendencies of the system are balanced. The lungs have a tendency to recoil to its deflated volume. The chest wall has a tendency to bow out. The point where the forces are neutral is the resting state. Pressure within the system is zero; the volume is called the functional residual capacity(FRC). We are interested in the pulmonary compliance during a dynamic pressure situation. The only reliable starting point is the respiratory system at rest, at FRC. Unfortunately, few experiments measure this directly.

Airway resistance is also dependent upon lung volume. As lung volume increases, airway resistance falls in a non-linear manner. Resistance is at its lowest at maximum volume. The reciprocal of airway resistance is conductance, and this is linear in nature, increasing with volume. Airway resistance also depends upon the action of the bronchial smooth muscle, and bronchial mucosa. Resistance is increased with contraction of the smooth muscle and mucosal inflammation. Finally, airway resistance depends upon the density and viscosity of the gas breathed. Divers know that at great depths large pressures are required to breathe compressed air, and that these pressures are greatly reduced breathing a helium-oxygen mixture. Thus airway resistance is lower at altitude than at depths encountered in diving.

As you can see from this review, the behavior of the respiratory system varies according to surrounding condition.[69,102] It would be ideal to make all measurements under standardized conditions. In this manner, the key determinants of pulmonary toxicity could be determined and accurately modelled. It is difficult to accurately define these theoretical considerations during experiments designed to answer the operational questions posed. For this reason, assumptions are made, and are stipulated in the next section.

The Dynamic Overpressure.

The ability of the lungs to tolerate changes in pressure determines guidelines for clinical medicine, diving practices, and aviation practices.[106] In clinical medicine and in aviation medicine, positive pressure breathing equipment applies a positive pressure into the lungs, creating a pressure gradient between the alveolus and the atmosphere.[11,19,25,26,30,32,33,36,51,58,75,82] Usually, a constant pressure is applied to the respiratory system over a period of seconds to hours. During the application of this "static" pressure, there may be transient peaks of "dynamic" pressure greater than intended, which last for fractions of a second to seconds. A dramatic example of a dynamic over-pressure situation lasting fractions of a second occurs during the explosive decompression of a fighter cockpit. Decompression in diving or aviation creates a pressure gradient between the alveolus and the atmosphere when the ambient pressure drops below the alveolar pressure. The duration of the pressure gradient caused by decompression is usually on the order of fractions of seconds to several seconds, and is a dynamic pressure, Figure 1.

In order to develop a standard for tolerable dynamic pressure, the conditions must be stipulated. The population for which the limit is defined is the awake and alert military aviator. The air is assumed to be wet, at room temperature. The airway is free from internal and external obstruction. The chest wall is free moving. The phase of respiration at which the decompression occurs is resting at the functional residual capacity, FRC. In the absence of the peak pressure measured at the mouth, the conditions necessary to characterize the decompression are the initial and final pressures and the duration over which the decompression occurred. Pulmonary damage is defined by any objective measure of pulmonary damage, and must be measured in isolation from effects of hypoxia, ebullism and evolved gas. A useful variation on this limit is the supported dynamic pressure. In this case, the aviator is awake, alert, and wearing some life support equipment. Unfortunately this will vary according to the equipment used. In all instances, variations from these conditions will be stated where appropriate. The reasons for these conditions will be covered in depth in later sections.

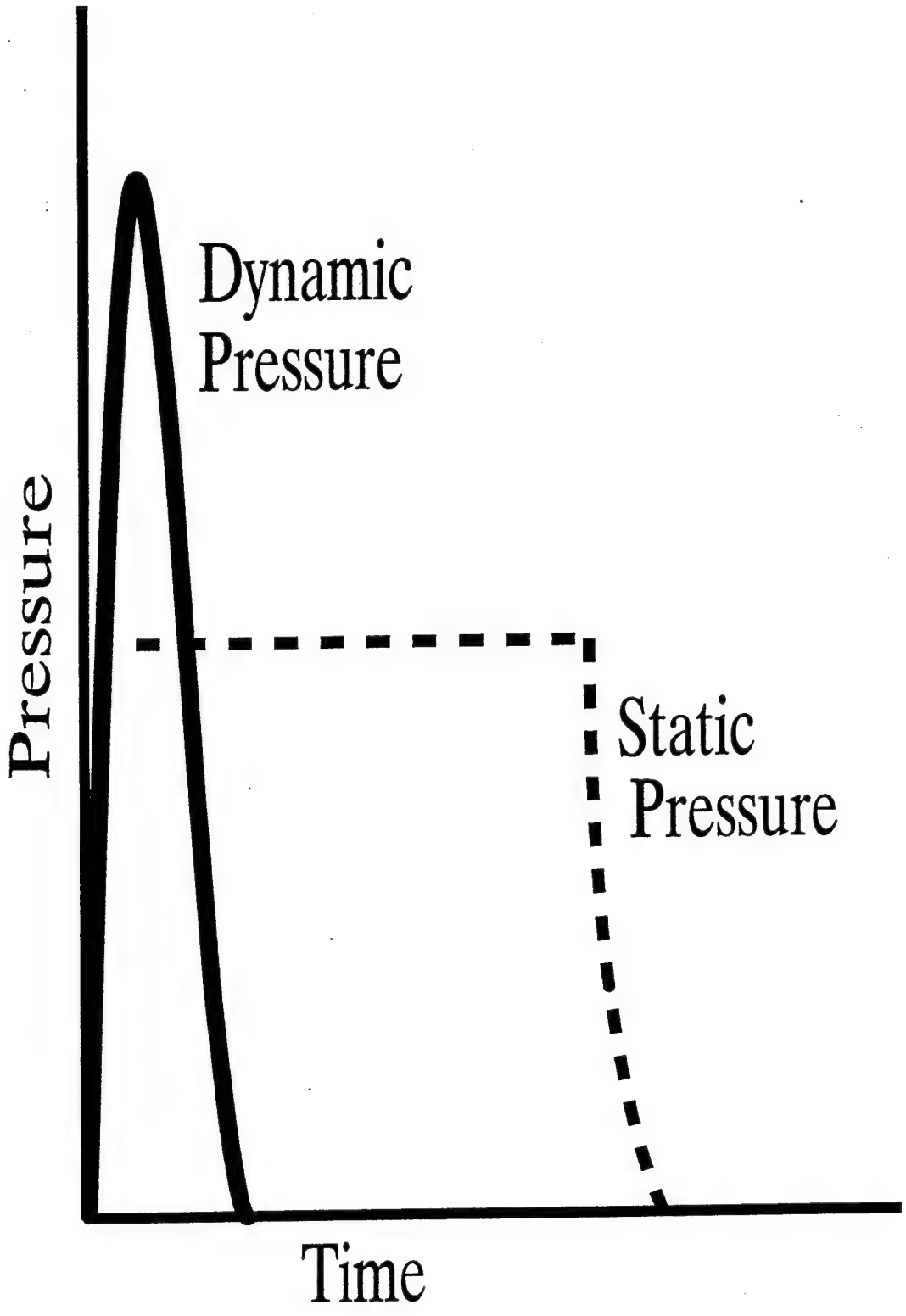


Figure 1. Diagramatic representations of positive dynamic and static pulmonary pressures over time.

The Process of Decompression.

Physics of Decompression.

At this point, it is necessary to review the theoretical concepts of decompression. Haber and Clamann discussed the theoretical basis of the physical process of decompression in 1953.[66] They propose a workable model of the process of decompression, divided into a time-constant which determines the time over which the process occurs, and a pressure function. The theoretical results are verifiable by experiment. It is useful to explore their summary.

Haber and Clamann chose not to differentiate rapid vs. explosive decompression as there has been no identification of a physical process which differentiates the two. Practically, however, the literature refers to rapid decompressions as those greater than one second in duration, and explosive decompressions less than one second. In this paper, the artificial distinction between rapid and explosive decompressions will be maintained. In effect, it is the point at which dynamic pressures could be separated from static pressures.

Rapid decompression occurring in an aircraft is not the same as a process occurring in a chamber. In the experimental situation, the gas expands into a large, but fixed chamber, contributing a rise in pressure to the chamber--a back pressure. In the aircraft, the environmental pressure stays constant. It is the back pressure created by the dynamics of the heat exchange, humidity and finite volumes involved that make the experimental decompressions different from the actual case. These differences may not be insignificant as the limits of human tolerance are explored, and should always be kept in mind.

In an ideal gas, the relationship of pressure, volume and temperature of a mole of gas are well described. Energy is not gained or lost in the system, and variation in any one variable will predictably change the others. In the experimental situation, heat is exchanged with the environment, and the ideal gas laws do not accurately predict the other variables. For example, energy is gained and lost in the process, a polytropic process. In a rapid decompression, temperature drops significantly. Changes in the realm of 100°C have been observed. In the experimental situation, heat is exchanged between the chamber and the environment, while in the aircraft, little additional heat is gained from the environment. During the first moments of the decompression, the temperature drops below the dew point, so the majority of the process occurs at 100% humidity. Heat is released from the condensing water, affecting the process as well.

The flow of a gas through an orifice depends upon the ratio of the cabin pressure to the ambient pressure, Pc/Pa. Flow through the orifice will reach the speed of sound in an ideal gas at a pressure ratio of 1.89 Pc/Pa. This is the critical pressure ratio. In an ideal gas, the flow will not increase in velocity even if the ratio is increased, a supercritical ratio. Stated another way, there is

a point at which gas cannot escape fast enough to equalize pressures across a newly created gradient. In the pilot at altitude in a decompression, this will appear to his lungs as a suddenly increased pressure within the pulmonary parenchyma, without chest wall support. In the polytropic process, like the experimental situation, the ratio at which the speed of sound is attained is always smaller than 1.89. This means that the limiting rate of escape of gas from the pilots lungs, and thus an apparent increase in pressure within the lungs, will occur when the ratio of the pressure in the lungs to the ambient pressure is less than 1.89. The rate at which a decompression occurs depends upon the orifice through which the pressure is equilibrated. All of the factors which interact at the orifice to determine the rate of the decompression can be combined into an expression in the units of time. Time of decompression is solely determined by the time factor in the equation, t_c, the time constant. Notice that it is independent of the pressure of the system.

$$t_c = \frac{V_c}{A \bullet c}$$

V_c is the volume of the cabin, A is the area of the opening and c is the speed of sound. Applied to the pilot at altitude, the volume is his lung volume, the area of the opening is determined by his upper airways, and the speed of sound is independent of the pilot. This equation states in a simplified manner the physical determinants of how fast a pilot can empty his lungs.

How long the pressure differential will remain is dependent upon the initial pressure differential. Pressure is figured into the equation as follows:

$$t_E = t_c \bullet P_1$$

P_1 is defined as:

$$P_1 = \frac{P_{co} - P_{ao}}{P_{co}}$$

P_1 does not depend on the absolute value of the pressure differential, but on the ratio of this differential to the initial pressure.

This is of course a simplified discussion, but it provides a basic understanding for the physical processes involved. Because this model is based upon rigid structures, it does not accurately predict the behavior of the human respiratory system. As applied to a human, it is only useful as a tool to understand the physical process of decompression.

The practical application of this is that one is able to determine times of decompressions if they are not provided in the experiment or the case report. However, rarely is enough information provided to complete this calculation. It is also a simple method the designer can use in determining decompression times for an aircraft cabin based upon failure of various components.

More complicated analysis and prediction of the environment during explosive decompression can be found. Topliff used mice to explore the mathematics of the lungs in regards to explosive decompression.[111] He found that if the decompression was rapid enough, then the pulmonary system could be considered a closed system, and the peak pressure that would be experienced could be calculated. Murphy and Engel,[95] and, Pardaens and Van De Woestijne[96] both explored a model of the pressure volume relationship of human bronchi. This is useful in predictions directed toward static pressures, but fails to address the types of stresses encountered in the dynamic over-pressure situation. Gagge and Sweeney developed a practical equation designed to help evaluate the danger of decompression in a particular aircraft.[59] They gave an equation which requires knowledge of the cabin volume and orifice causing the decompression. This is useful to the designer, but not for predicting stress on the human lung. Computer models of external blast injury as represented by Stuhmiller et. al. more closely approach the reality of a decompression.[109] Roth, in 1968, summarized these concepts as applied to the human pulmonary over-pressure issue.[101] However, there is no data correlating lung injury directly with impulse. Translating blast wave data is not a good model for the lung in relation to the over-pressure problem of aircraft decompression as it is orders of magnitude higher in pressure and briefer in duration.[28,99]

In summary, be aware that the theoretical literature exists, but does not yet assist us in the practical question of "Will Captain Jones survive this explosive decompression?".

The conclusions to be drawn from the models of decompression are:

1) The maximal possible amplitude of the transmural pressure in the lung model is equal to the pressure difference of decompression (Pi-Pf).
2) The fraction of the total pressure difference effective in the lung is dependent on the V/A ratio in the lung to that of the suit or cabin. In other words, consider the differential between the lungs and the cabin pressure first, especially if the cabin decompression is prolonged.
3) The pressure ratio of decompression Pi/Pf determines the force times time integral or impulse for any given amplitude of the transthoracic pressure transient, and therefore the duration of a critical over-pressure. It is assumed that the impulse could be associated with the degree of damage sustained. This, it turns out, is extremely difficult to isolate in a biological system.

Human Decompression.

Observation of the thorax during decompression provides some insight into the actual performance of the human body during a decompression. According to cinematographic data, decompression of the lungs takes place in three phases.[65]

Phase one occurs under essentially isometric conditions with no change in volume, owing to the inertia of the system. The highest transthoracic pressures are probably attained during this phase. This is the phase where the peak mask pressure will be identified, and will be the peak dynamic pressure. This is the phase where it is proposed shearing of pulmonary parenchyma occurs similar to blast injury.

In the second phase, the pressure is attenuated due to expansion of the chest and also to the continuing escape of gas through the airways. Structural damage is conceivable when the peak pressures create powerful dynamic forces opposed by the inertia of the system. Due to differences in densities, differences in acceleration under impulsive pressure loading could result in shearing and spalling lesions similar to those encountered during blast. During normal respiration, the bronchial tree expands uniformly in all directions. With rapid over-expansion, peribronchial alveoli are torn away from nearby interstitial tissue, simultaneously tearing alveoli and small veins.[44,65] Lesions attributable to pressure damage are small vessel rupture and petechial hemorrhages, emphysematous changes or alveolar septal rupture and cellular fragmentation and disruption.[65,70,74,112] Alveolar air is allowed to escape into the torn veins held open by elastic recoil of surrounding tissue, and along the peribronchial interstitial routes to the mediastinum.[56,91] Tearing of the pleura may occur with air escaping into the pleural potential space.

In the third phase of maximal expansion, the conditions are again isometric until the over-pressure is dissipated and the lung volume decreases. During this phase, the mechanism of presumed injury is the rupture of tissues at limits of their tensile strength. Penetration of gas bubbles into the bloodstream can most likely take place when the lungs are fully expanded and a high gradient is created between the intrapulmonic pressure and that in the pulmonary veins and the left atrium. This is the phase in which chest counter-pressure is used to limit damage sustained.

Physiologic pressures measured during valsalva approach 60-100 mmHg.[113] Pressures can be higher during defecation, parturition, coughing and weight lifting.[74] These physiologic actions rarely cause barotrauma to the lungs because of the manner in which they differ from the decompression scenario. During these physiologic actions, the musculature of the chest wall, diaphragm and abdomen contract in concert to support the pulmonary parenchyma, increasing pressure by reducing volume. Pressure is equally distributed over the lung, and over-distension does not occur. During explosive decompression, the chest wall does not provide adequate support for the intrathoracic pressure, and allows the lung to enlarge past their maximal volume in an attempt to reduce the pressure.

This model is consistent with our knowledge of probable mechanisms of pulmonary damage during over-pressure events, and provides a framework for understanding the issues involved. In a later section, we will explore some of the confounders in over-pressure research. Next we will review the current over-pressure guidelines.

The Current Pulmonary Pressure Guidelines.

Most readers are familiar with the static limits of 80 mmHg pressure for a human with an unsupported chest wall,[9,77,97] and 190 mmHg for a supported chest wall.[50,73,74,106] The static unsupported chest wall limit has been passed through the literature since the 1930's. It is again useful to go back to the original papers to gain an understanding of where the limit came from, and the strength of the data. Polak and Adams, 1932,[98] are cited often as having written the first paper promoting the 80 mmHg limit for safety. Their paper was written in response to sudden deaths of naval personnel which occurred immediately after ascent in emergency escape training for submarines. They performed an initial series of experiments on rats where the etiology of death arising from ascent to the surface was explored. It took an original experimental design to demonstrate the air emboli in the carotid circulation of dogs. Pressures of less than 80 mmHg did not usually cause embolism, while pressures greater than 90 mmHg usually caused emboli.

In another group of experiments, they addressed the issue that pressures at depth were routinely greater than 60 mmHg and people did not routinely die, so the issue of pressure with and without pulmonary distension was explored. Dogs were used, the chest and abdomen were splinted to prevent expansion past that of deep inspiration, one animal per experiment. They drew the conclusion that pressure plus chest distension was responsible for air embolism in a single animal cross-over experimental design.

This paper established the etiology of air embolism as a pulmonary overpressure and distension phenomena. It did not report experimental results on human subjects, or seek to prove or establish a range of pressures or norm for maximum safe pressures. It concluded with a discussion of the medical management of such cases. Prevention was to occur by proper training. The authors were often misrepresented as having established a safety limit for decompressions.

In a similar manner, Benhke, 1933, is cited.[23] He discussed several fatal cases of air emboli in submarine escape training. Cases were anecdotally presented, and much of the conclusions were conjecture. There is no firm data presented with which to draw data from on the safe limits of pulmonary pressure.

Experimental work looking at peak static pressures causing pulmonary pathology has also been cited as substantiation for a dynamic overpressure limit. In the most well known of these studies fresh, unchilled human cadavers were used to determine pulmonary pressure limits. Malhoutra and Wright, in 1961,[90] set out to determine roughly the static pressure limits of the lungs and the

effects of binding the chest and abdomen on peak pressure tolerated. Only five bodies were available for use, from 27 to 64 yrs of age. Known volumes of air at atmospheric pressure and 18° C were injected into the trachea by a mercury pump. Intratracheal pressure was continuously monitored. Air was pumped at a rate of 30 ml per second. After 200 ml, the pump was stopped, and the pressure tracing was observed for loss in pressure which would indicate a breakdown in the pulmonary tissue. A summary of the cases is presented in table 1.

TABLE 1: MALHOUTRA AND WRIGHT, 1961,[90] DATA SUMMARY

Case	Sex	Age	Binding*	Pressure (mmHg)	Volume (Liters)
1	Female	47	None	93	6.3
2	Male	64	A	80	5.8
3	Female	27	A, C	190	6.2
4	Male	61	A, C	133	7.6
5	Male	62	A, C	189	9.8

*A=Abdomen, C=Chest.

A brief review of the pathology follows. Case 1's right lung developed a pneumothorax near the site of pulmonary adhesions located in the basilar sections. Both lungs also had diaphragmatic adhesions, not associated with pleural tearing. Case 2's gross examination revealed multiple large bullae over the anterior surface of the right lung, and along the anterior medial border of the left basal lobe. Pulmonary adhesions were found along the dorsal border of the left lung, but were not associated with tearing. No pneumothorax was identified, but interstitial emphysema was significant enough to release the pressure applied to the pulmonary system. Case 3's lungs had marked interstitial emphysema, some petechial hemorrhage, but no pneumothorax. Case 4's lungs revealed considerable interstitial emphysema. Adhesions on the right lower lobe to the diaphragm did not cause pleural tearing. Emphysematous blebs on the dorsal surface of the right lower lobe did not rupture. Case 5's lungs had marked interstitial emphysema, multiple emphysematous bullae, no adhesions, no pneumothorax.

It is clear that this study had several limitations. The subjects of the experiment were not representative of the population of interest, the active military aviator, and in general were advanced in age. In addition, statistical analysis was not attempted due to the small sample size. The authors were aware of this, but were limited by the availability of cadavers which were thought to have pulmonary systems unaffected by the cause of death, or pulmonary systems that were the cause of death. The experiment did not readily detect the subtle release of pressure present as subcutaneous or mediastinal emphysema. Indeed the lung volumes on cases 4 and 5 were quite large; some of this volume was found in the mediastinal and subcutaneous chest

tissues. Finally, the study could not account for the differences between living and dead tissues. In the living subject, diaphragmatic and intercostal muscle tonus may affect the failure pressure. Safe intratracheal pressure is much less in anesthetized animals than in animals without muscle relaxants. Those with relaxants develop emphysema around 40-60 mmHg while those without need 60 - 100 mmHg.[90] On a molecular level, early degradation of proteins, collagen, and alveolar tissue may serve to weaken the pulmonary system enough to lower the peak pressures sustained.

This study proves that chest and abdominal binding for prevention of pulmonary over-expansion does allow greater pressures to be sustained. It demonstrates that pulmonary pathology such as adhesions can affect the ability to tolerate high intrapulmonary pressures. It also provides gross evidence of the movement of air from the alveolus into interstitial tissue planes and further into the mediastinum. There is evidence here that this process begins at around 60-100 mmHg in both bound and unbound cadavers. The authors conclude that over-expansion of the lungs is the cause of pulmonary barotrauma. It cites the peak static pressure limit in unsupported humans as 80 mmHg, and in supported at about 190 mmHg. These are reasonable numbers guiding an investigator to approach pressures above these with caution and adequate safety measures.

In 1958, Schaeffer et. al. explored the effects of slow pressure changes during decompression from depths of 100 to 200 feet equivalent depths of water.[104] These experiments helped lead to an early understanding of the etiology of pulmonary pathology, and helped to confirm previous limits set for pulmonary over-pressure. Using dogs, the authors explored the pressures required to produce air embolization and the effects of thoracic and abdominal binding.

The study provided data curves of pressures of relevant pulmonary and cardiac structures when exposed to pressure changes with and without binding. With binding, arterial pressure remains relatively constant, and intra-tracheal pressures rise rapidly. Without binding, arterial pressure falls, intra-tracheal pressures rise slowly. Transpulmonic pressure gradients less than 60 mm Hg and transatrial pressures less than 50 mmHg did not produce any air embolism in dogs--even when intratracheal pressures reached excess of 90 mmHg.

This work confirms the Polak and Adams[98] work that the critical intra-tracheal pressure is approximately 80 mmHg. Binding prevents over-distension of the lung. High intratracheal pressure can be withstood--180 mmHg as long as the transpulmonic pressure does not exceed the critical level of approximately 60 mmHg. It reaffirms disease processes may result in gas entrapment in small portions of the lung.

In comparison to studies done at altitude, these pressure differentials occur over long periods of time, 1-2 minutes. The pressure range is much greater, many atmospheres, at high final pressures, versus fractions of atmospheres at low initial and low final pressures.[20] It is similar in the manner that the entire body is exposed to the pressure change and the damage is caused by the delay in equilibration of the gas in various portions of the body--with only the gasses in the thorax producing fatality.[21]

It is clear now that our currently understood limits of pulmonary over-pressure are relevant to a more easily explored environment of slow decompressions in a diving environment. While these limits are useful guidelines, they do not completely or adequately predict the performance of man at altitude in a rapid or explosive decompression environment.[42] For this reason, a review of the literature was undertaken to identify data which might provide guidance in the realm of the explosive decompression environment.

METHODS

All literature searches were conducted through the Strughold Library, Armstrong Laboratory, Brooks AFB, TX. Initial searches were conducted through MEDLINE and Defense Technical Information Center (DTIC). MEDLINE and DTIC searches were conducted for the same search terms. Search terms used were: lung over-pressurization, pulmonary over-pressurization, over-pressurization, over-pressure, air embolism, decompression sickness, embolism, gas embolism, lung, lungs/ventilation system, pneumothorax, pneumothorax(truncated), breathing gas, cabin pressure, cabin pressurization, pressurized cabin, decompress, decompression, explosive decompression, rapid decompression, oxygen, sonic decompression, ventilation, assisted positive pressure breathing, positive pressure breathing, pressure breathing, altitude pressure breathing, positive pressure breathing for G, COMBAT EDGE, partial pressure suit, pressure suit, altitude, altitude limit, emergency pressure suit, high altitude, limits, maximum altitude, barotrauma, iatrogenic pneumothorax, ventilator over-pressure and aviation medicine. Printouts of searches contained titles and most had abstracts. Articles were selected for inclusion in the review based upon at least one of the following criteria: presentation of original research with data from human or animal models directly related to pulmonary over-pressure; review article with summary of data or research questions answered to date; bibliography; presentation of human respiratory performance limit, or original model or hypothesis concerning pulmonary over-pressure. These guidelines were meant to be very broad initially, to encompass all of the possible articles on the subject. Approximately four hundred articles were identified by this search. A secondary search was conducted beginning with selected textbook bibliographies from: Aviation Medicine, Second Edition, John Ernsting and Peter King[106]; Fundamentals of Aerospace Medicine, Roy DeHart.[73] In addition, bibliographies from AGARD publications in Aerospace medicine, No. 312,[2] 322[4] and 516[3], and the Bibliography on Aviation Medicine, Volume I[6] were also used as a starting point for the secondary search. References cited in the bibliographies relating to any of the four criteria listed above were obtained. These bibliographies were also searched. As long as the bibliography search produced new articles, the search was continued. This yielded 98 articles previously identified in the DTIC and MEDLINE searches, and 25 not previously identified. Those not previously identified were primarily in foreign journals, and were prior to 1956. Articles which provided human or animal experimental data were searched by author using Citations Index for 20 years after publication. In an attempt to identify works done by the same author that produced data on pulmonary over-pressure, the citation index was used to

find articles for the 20 years preceding publication of the original article. No new data sources were identified in this manner. Finally, the Armstrong Laboratory Hypobaric Decompression Sickness Research was manually reviewed. No new sources were identified.

The earliest identified source of experimental information was Robert Boyle's work in 1670.[29] No references have been identified after 31 Dec 93.

Sweeney's 1944 work published in *Air Bulletin* 1:1-10 was extensively cited.[110] Unfortunately I have been unable to locate a copy of the original article. Data presented by Sweeney was provided in Fryer's work.[55]

French language articles were translated with the assistance of the Strughold library.[65,115,116]

It will be apparent that with a few exceptions, the diving medicine literature is not extensively cited. While the diving physiologist and dive medical officer have a need to understand pulmonary pressure limits, the diving experience is not directly comparable to explosive decompression.[20,21,42]

An additional body of information is contained in isolated case reports.[13,38,40,72] However, fatal exposures at altitude are usually accompanied by more speculation than fact. The lack of accurate data prevented the use of any of these data points.[54] Additionally, data is missing from operationally active military flight squadrons. Some data has been collected for the COMBAT EDGE program, but has not been compiled and published in any manner. It is critical that any exposures with accurate information obtained from flight data recorders be analyzed and published if the data is found to present a unique exposure to a pilot.[13]

Analysis of the articles was accomplished by an initial survey review of the articles. Publications which contained original data from animal and human subjects were noted and separated from the other publications. Only those publications which provided direct support or additional information relevant to the analysis were retained, and cited in this work.

Data analysis was conducted by identification of the basic elements needed to determine the nature of the decompression. Information required to adequately describe the decompression is: the initial and final atmospheric pressure, and the time allotted for the pressure change. All times were given in units of seconds. In cases where time ranges were given, the time for the pressure change was assigned the longest duration possible in the experimental conditions. Pressures were reported in either pounds per square inch (PSI), millimeters of mercury (mmHg), or altitude equivalents (feet above sea level). Pressures were converted to mmHg using the United States Standard Atmospheric Pressure Table 1[10] when reported in PSI or altitude equivalents. Where pressure ranges were given, the pressure ranges giving the smaller pressure changes were used. In some cases, initial and final pressures were not given, but the magnitude of pressure changes was recorded. Data used to produce graphs are included in the appendix. Where possible, data was summarized without loss of important detail. It is not possible at this time to foresee all of

the possible scenarios which could place humans in dynamic over-pressure environments. It is the objective of this work to provide a summary of all available objective data relating to the limit of human tolerance to pulmonary over-pressure within the currently defined realm of aviation medicine, not to provide a summary of the applications of this limit.

RESULTS

The maximum pressure that can be safely tolerated by the human pulmonary system in a dynamic over-pressure situation is unknown. This measurement has not been performed. Evidence will be presented which suggests that the unsupported chest wall of the human population can safely support 80 mmHg static and dynamic over-pressure of the lungs. This is not to be misunderstood as an absolute limit for the population. It is likely that the pilot population of the United States armed forces could tolerate higher dynamic pressure, due to a lower prevalence of pulmonary pathology than in the general population.[5,117] (Individuals with pulmonary pathology are selected against in the pilot selection process.)[117] Safe static pressure in the human population wearing chest and abdomen support devices is at least 190 mmHg. It is probable that pilots wearing well designed life support equipment could endure higher pressure without permanent injury. The problem lies in the lack of human data, partly due to the fact that humans have never expected to be exposed to decompressions in excess of the data presented in this summary.

The range of decompressions tolerated by human subjects, and documented in the literature reviewed is presented in Graph 1, Human Decompressions, Pressure Range vs Time. Each bar represents, at minimum, one safe decompression. These represent the decompressions most commonly employed in experimental protocols. They do not include decompressions with humans in pressure suits. There are anecdotes of more extreme decompressions, but were not published, and are not included. A summary of the data points for the graph is listed in the Appendix. Decompressions longer than 5 seconds in duration were not included as these were not in the realm of explosive decompressions.

Graphs 2 and 3 show areas of relative safety and uncertainty. Graph 2, Human Decompressions, Pressure change vs Time, presents the same data seen in Graph 1, but uses only the absolute pressure difference on the y axis. This does not account for differences in the decompression due to density effects and effects at altitude. The "Zone of Certainty" represents human decompressions accomplished safely. The "Zone of Uncertainty" depicts decompression environments unexplored with human subjects. There are no experimental decompressions less than 0.01 second in duration. Safe decompressions can be bounded by pressure changes less than 100 mmHg in 0.01 second, 375 mmHg in 0.1 second, and 675 mmHg in 2 seconds. Beyond these points, published data was not identified.

Graph 3, Human Decompressions, Relative Gas Expansion vs Time, again plots essentially the same data. The equation used is:

$$RGE = \frac{P_{INITIAL} - 47}{P_{FINAL} - 47}$$

$P_{INITIAL}$ is the initial pressure, and P_{FINAL} is the final pressure of the decompression. It accounts for the effects of water vapor at body temperature during the decompression. It is not useful for decompressions with a final altitude approaching 47 mmHg. It is a more traditional way of representing the data, but does not significantly add to the simpler graph of Pressure Change vs Time. Decompressions measured in terms of relative gas expansion are bounded by decompressions less than 2 in 0.01 second, 15 in 1 second and 18 in 2 seconds. The RGE term has little meaning past 20, due to mathematical considerations.

Results of animal experiments are graphed on Graphs 4-6, Animal Decompressions. Limits of human decompression experience have been superimposed for comparative purposes. The most significant data is summarized on the graphs; duplicate data points were excluded for clarity. The data indicate decompression without serious temporary or permanent effects from as brief as .01 sec to 2 sec over a wide range of pressure changes. All three graphs demonstrate limits of safe animal decompressions. In general, decompressions less than 0.01 second are not well explored, and appear to be unsafe over large pressure changes, Graphs 5 and 6. Changes of pressure of one atmosphere in less than one second can cause death, but may be tolerated in a large percentage of the population. Changes of one atmosphere in greater than one second are generally tolerated by animals and are not presented except in graph 1. These graphs generally show that human data are less extreme than the animal data.

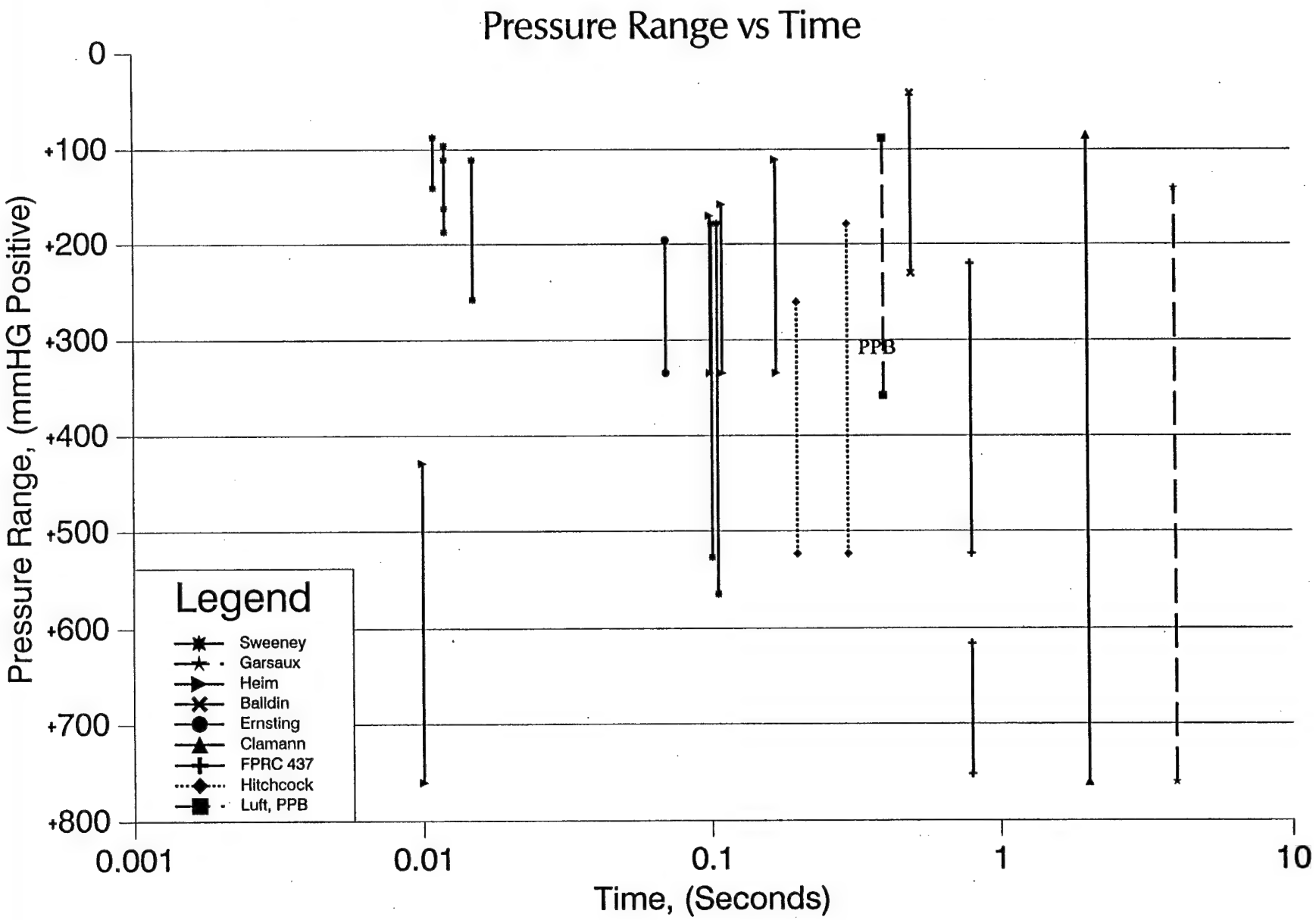
There are limits to the animal data which could be used to guide future human experimental work. Hall, 1957, definitively demonstrated that there are definable limits to tolerable dynamic over-pressure exposure.[67] These experiments are connected point to point to form the "Limit of Animal Safe Decompressions." Pulmonary toxicity occurs below this line, as this approximates a lethal exposure to 50% of the animals exposed. Lethal exposure provides a more readily determined endpoint of toxicity in animals than does quantification of pulmonary hemorrhage. That is why this line can be drawn as a relatively firm limit of exposure.

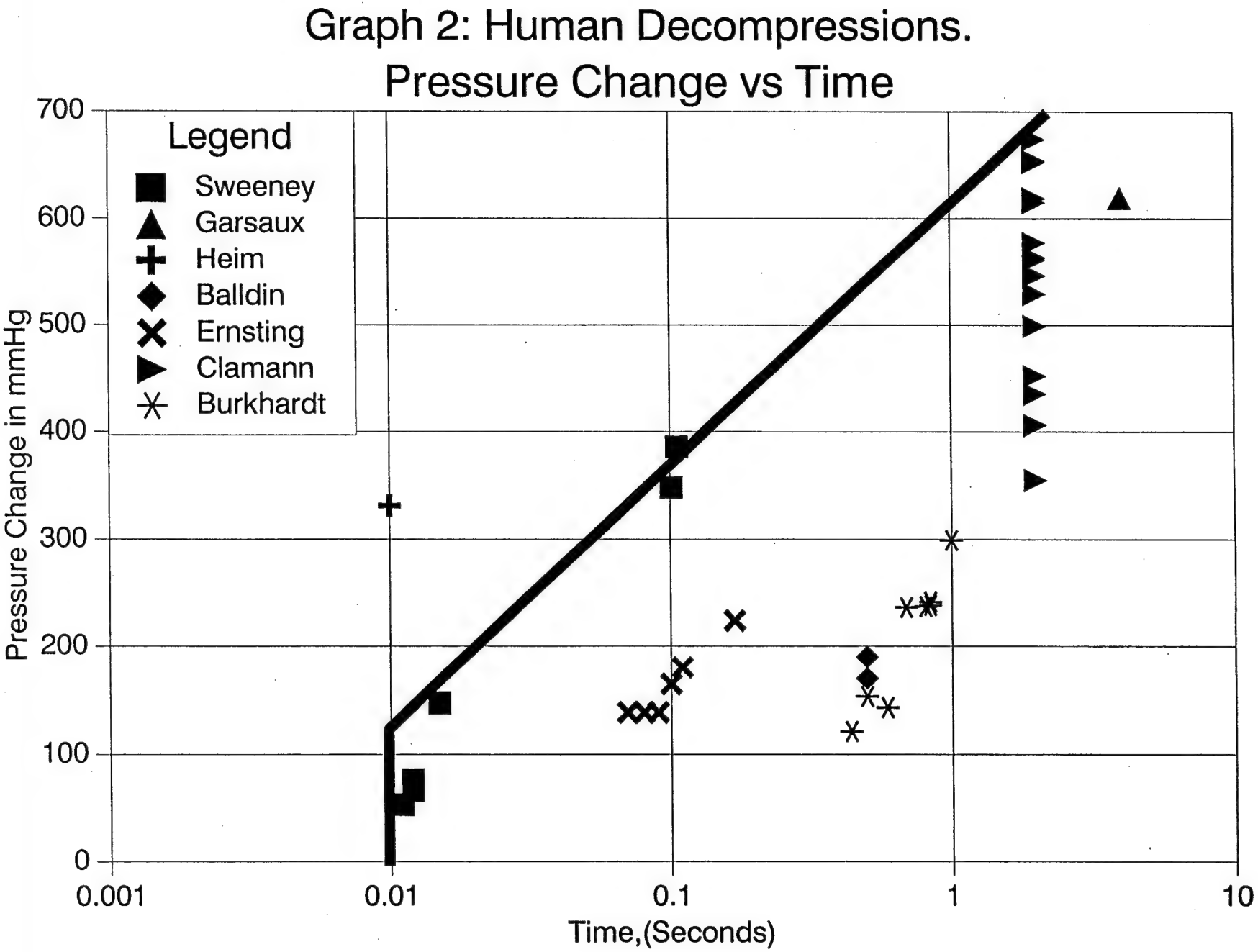
The validity of animal models for estimating tolerance limits for human pulmonary toxicity is likely good. In the only study which evaluates the static and static supported pressure limits of a large variety of animals, J.P. Henry, in 1945 determined the "strength of the alveolar wall was of the same order as that of the capillary bed, namely 50-100 mmHg."[74] The animals varied in size from mice to steer. The primary difference was in the rigidity of the thorax. Dogs and steer performed most like the cadavers in the Malhoutra study.[90] The flexibility of the chest wall in cats, rats and mice limited their applicability to the human situation. These smaller animals will tend to underestimate the human tolerance for a given static pressure situation.

In summary, the safe limits for static pulmonary pressure are conservatively set at 60-100 mmHg, and for supported static pressures at 170-190 mmHg. These limits do not account for dynamic

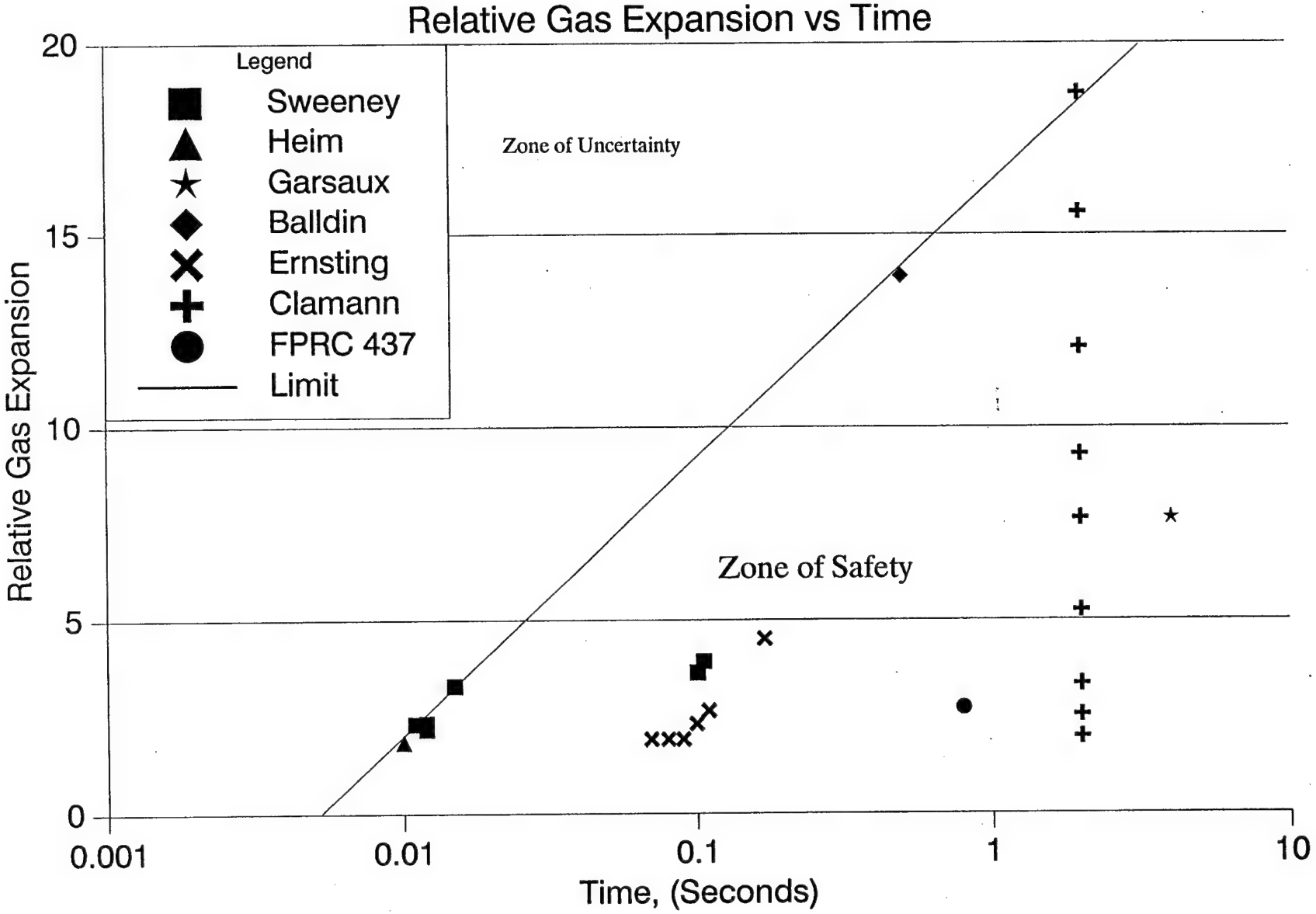
over-pressure situations. Animal evidence suggests that greater pressure could be tolerated in humans with minimal toxicity. Pilots will be exposed to pressure beyond the currently accepted safety limits, and most will survive. It will be the responsibility of flight surgeons and physiologists to document and publish these events in order to adjust our current understanding to improve the capability of future aircraft.

Graph 1: Human Decompressions

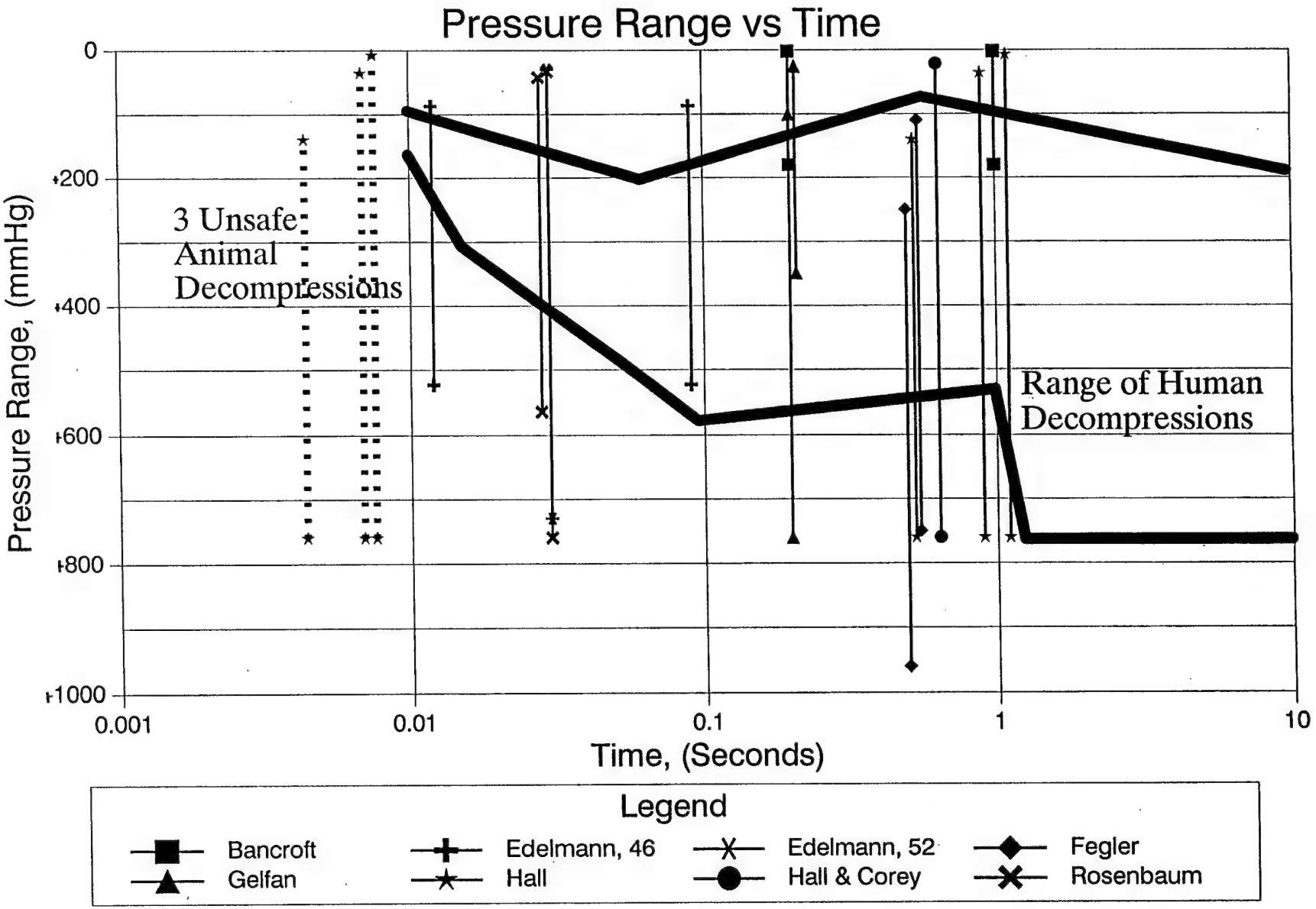



Graph 2: Human Decompressions.
Pressure Change vs Time
Legend
Sweeney
Garsaux
Heim
Balldin
Ernsting
Clamann
Burkhardt
Pressure Change in mmHg
700
600
500
400
300
200
100
0
0.001
0.01
0.1
1
10
Time,(Seconds)

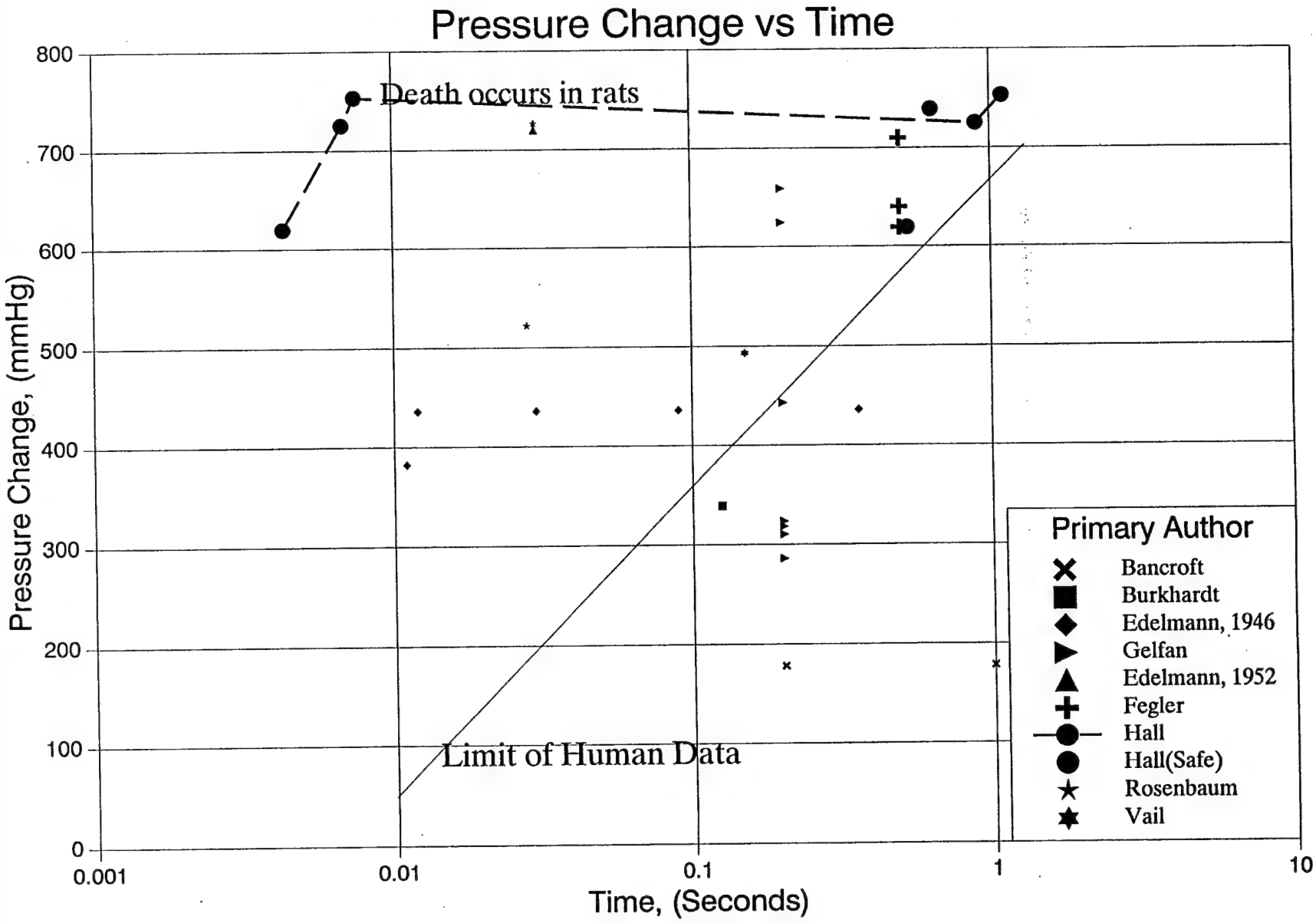
Graph 3: Human Decompressions.



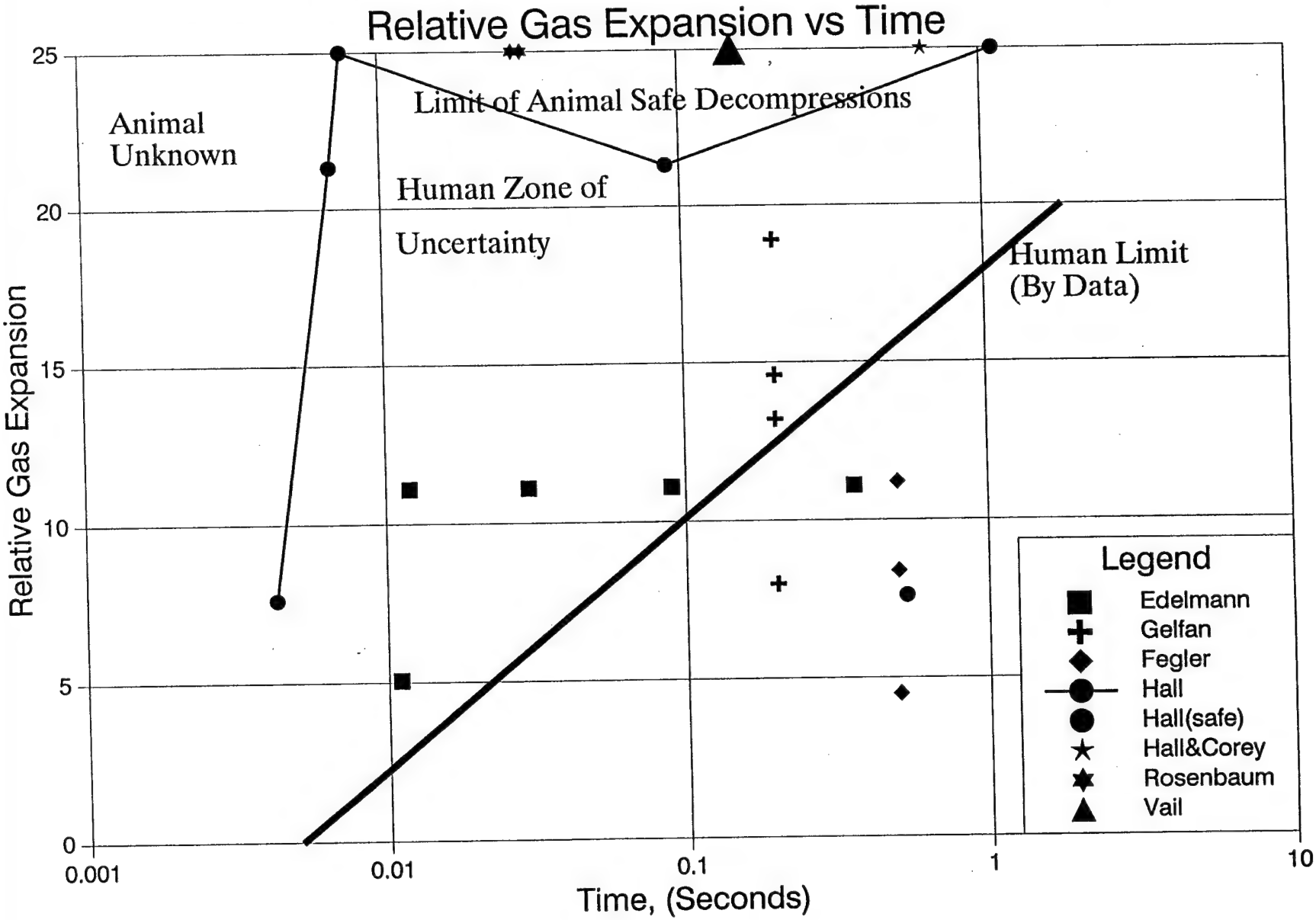
Graph 4: Animal Decompressions.



Graph 5: Animal Decompressions



Graph 6: Animal Explosive Decompressions



RESEARCH PROBLEMS

Investigation of the isolated effects of pulmonary over-pressure is confounded by the effects of hypoxia,[17,39] ebullism and evolved gas phenomena--decompressions sickness.[37] It is complicated by the variability of the biological system under study, including alterations from normal, induced by disease processes. Poor study design has also plagued research in this area.[94]

Like all tissue, the lung displays its response to a wide variety of insults in limited ways. Acute insult such as decompression, hypoxia, decompression sickness and ebullism damage the microvasculature.[44] Whatever the mechanism, the hemorrhage, fluid filled alveoli and inflammatory response appear identical in the pathologist's section.

Clinically significant decompression usually lowers the ambient pressure enough to reduce the oxygen available to the lung.[93] When the oxygen partial pressure is reduced below the minimum required to sustain cellular function, hypoxia results. A complete lack of oxygen is termed anoxia, and is just the most severe state of hypoxia.

Hall and Corey, in 1950, correctly identified hypoxia as cause of injury in explosive decompression.[68] Their purpose was to elaborate the duration of anoxia at altitude required to cause explosive decompression injury. Rats were explosively decompressed from sea level (760 mmHg) to 80,000 feet (21 mmHg) in 0.64 sec. Also, animals were decompressed explosively and then recompressed almost as rapidly. 20 rats remained at altitude for less than one second, after decompression from sea level (760 mmHg) to 80,000 feet (21 mmHg) and were the control animals. They all survived the decompression without apparent injury, but no histologic or gross pathology evaluation was performed. When time at altitude was prolonged beyond 10 seconds, deaths began to occur. At 40 seconds, 100% of the rats died. When the thorax was taped to permit only minimal respiratory activity, 8 of 10 rats survived a 40 second exposure to 21 mmHg (80,000 feet). The lesions observed in the rats that died at altitude were identical to the lesions observed in rats exposed at sea level to 100% nitrogen. Rats were cooled to reduce oxygen consumption, and exposed to 80 seconds at altitude without fatality. When treated with thyroxin to increase oxygen consumption, all rats died in 20 seconds. When the animals were splinted during the explosive decompression, 8/10 survived for 40 seconds.

The study concluded that anoxia was the major cause of injury from explosive decompression. The authors were unable to explain the reason that binding the thorax allows survival through an otherwise fatal exposure. They suggest that the rat thorax is more flexible than the human thorax, and thus is an experimental result not applicable to humans. Hall and Corey were on track with their hypothesis that hypoxia could cause the same injury seen in explosive decompression, but incorrectly concluded that the flexibility of the chest wall was not a factor in humans.

Fegler, in 1941, published good evidence that some of the pulmonary damage seen during decompression was due to hypoxia.[53] The primary objective of the experiment described was to characterize the physiologic response of guinea pigs to explosive decompression and anoxia. The basic experimental design was to expose guinea pigs to decompression while varying the partial pressure of oxygen, attempting to separate the effects of the pressure change from the effects of anoxia. Sixteen animals were explosively decompressed (in about 0.5 seconds) from 960-1160 mmHg to 250 mmHg, with a minimum pressure change of 710 mmHg. The atmosphere was 100% oxygen. All survived and none demonstrated acute pulmonary change by histology. In a second series of experiments, final pressure varied slightly, but oxygen pretreatment and oxygen at altitude were varied. The decompression went from sea level (760 mmHg) to 41-45,000 feet (110 mmHg). Pulmonary lesions worsened with decreasing exposure to oxygen. The animals enduring a 710 mmHg pressure change while maintaining a normal oxygen partial pressure were uninjured, while animals experiencing a 650 mmHg change with exposure to low oxygen partial pressures experienced injury. The authors concluded that the pulmonary lesions were mainly due to anoxia, and not pressure change.

Decompression sickness can lead to pulmonary pathology independent of the effects of rapid decompression.[37,44,89] The primary mechanism of damage is the obstruction of the microvasculature with bubbles formed from gas dissolved in the tissues.[23,37,114] Bubbles can form in the interstitial spaces as well. Denitrogenation appears to reduce the damage to the lungs from the decompression. Short duration exposure to low pressure, less than 90 seconds, resulted only in minor trauma. The combined effects of mechanical stress from the enlarging bubbles and their resultant obstruction of oxygen delivery leads to tissue damage. This is a potential confounding effect, but was not seen in these studies as most of the protocols returned the subject to the original altitude within seconds of the decompression.

Ebullism combines the effects of hypoxia with evolved gas problems. Ebullism occurs in humans when the ambient pressure drops below 47 mmHg (63,000 feet), the vapor pressure of water at 37° C. At pressure below 47 mmHg, the water in the lungs vaporizes, and gasses in solution, oxygen, carbon dioxide and nitrogen all exit. Experiments with terminal pressure lower than 47 mmHg will all expose the lung to damaging forces other than just the decompression.[46]

Kemph et. al. published a series of observations describing decompression to 30 mmHg (72,000 feet), with the purpose of describing survival time at altitude without oxygen.[81] The experiments were identical in rate and magnitude of decompression to the study of Edelmann and Hitchcock, 1952.[46] Most significant were the x-ray and fluoroscopic observations made on dogs at 30 mmHg ambient pressure. There was gas formation in all of the potential spaces, such as the peritoneal, pleural, joint cavities and along fascial planes. Vail confirmed these findings, and presented evidence for vapothorax on x-ray and fluoroscopic studies of animals in ebullism conditions.[113]

Further observations of animals under ebullism conditions were made by Bancroft and Dunn, in 1965.[18] They set out a series of controlled experiments to determine time of consciousness and survival in animals explosively decompressed from 35,000 feet (180 mmHg of Oxygen) to 1 to 2

mmHg (150,000 feet) in 0.2 to 1 second. The primary model used was dog, with 126 animals used in the entire experiment. The purpose of the study was to determine onset of incapacitation and survival at very low pressure, 1-2 mmHg, after rapid decompression. Previous experiments had explored the pressure region of greater than 30 mmHg (72,000 feet), but none had explored near vacuum conditions. Within seconds of exposure to 1-2 mmHg (150,000 ft) the animals showed marked evidence of gas expansion and water vapor evolvement, with expulsion of gas from the bowel and bladder. The animals became completely immobilized, with the skin inflated like a goatskin bag. Upon recompression, the animals deflated dramatically to their normal appearance. At 45-50 mmHg, a major portion of the deflation was complete, suggesting that water vapor was the predominant gas involved in the distension of the animals.

Animals that were exposed to near vacuum conditions for less than 120 seconds survived, though with some pulmonary damage. Pulmonary pathology could not be solely attributed to barotrauma, due to the presence of anoxia and evolved gases (both decompression and ebullism) as part of the experimental design. In addition, the anoxia caused some generalized muscle spasticity, convulsive seizures, apnea and gross swelling of tissues. All of these actions may also contribute to pulmonary damage seen on autopsy. It is important to note all animals undergoing rapid decompression while breathing 100% oxygen and immediately recompressed on oxygen survived. There was a marked rise in mortality when animals were recompressed on room air. The authors demonstrated that extensive hemorrhage, pulmonary edema and atelectasis are more related to degree of anoxia than to the rapid pressure changes. It is reasonable to assume that some pulmonary pathology will result from defacto exposure to the near vacuum.

Finally, problems are encountered with the subjects chosen for the experiments, and faulty experimental design. Two studies which provided animal data were compromised by ill animals. Edelmann et. al., 1946, used dogs that were all sick with parasites, one had distemper.[45] Gelfan's 1951 study was seriously compromised by ill animals.[61] Varying degrees of pulmonary tuberculosis (TB) were found in 75% of the monkeys. In addition to TB, "A good number of the animals" were infested with parasites, mainly *Oesphagostomum brumpti*, some heavily. Monkeys died in the laboratory from these and other diseases before any experiments were performed. Not only did these illnesses make determination of survival curves difficult, it made determination of pulmonary damage due to decompression impossible. 45% of the animals that underwent autopsy had no pulmonary lesions, although half of these had pulmonary TB. The remaining 55% autopsied had hemorrhagic lesions and areas of atelectasis. Identical hemorrhagic lesions were also noted in a monkey that had not been decompressed, but had TB. Because pulmonary TB was not controlled, it became difficult to relate the decompression exposure to pulmonary pathology found on gross exam.

Edelmann et. al. had problems not only with ill animals, but their study also suffered from poor experimental technique.[45] Some of the animals were anesthetized with nembutal during the decompression; some were not. The animals were then sacrificed with a variety of techniques; intracardiac $MgSO_4$, nembutol, or electrocution in one group, and nembutol and intentional pneumothorax in another group. It is not clear why various methods of sacrifice were chosen,

although these techniques were standard in the experimental literature of the era. However, because the methods of sacrifice were uncontrolled, and some had greater possibility of causing pulmonary pathology, it is difficult to assign the pulmonary damage found to the decompression. Gelfan, 1951, did not have a control population of animals.[61] Animals had serious infections which resulted in deaths during and after the decompression. He had 17 unintentional deaths in the study. Some animals died from the altitude exposure in the chamber, some died from nembutal overdoses, aspiration, and of course infection. Without a control population, he was unable to determine the effects of the decompression exposures on mortality.

Disease processes and biologic variability will predispose some subjects to damage at pressures lower than expected. This was seen in the animal experiments where the animals were ill with pulmonary TB and parasitic infestation. Any clinical condition predisposing to spontaneous pneumothorax will exacerbate the damage.[56,101] Congenital cysts, or post-infectious or asthmatic bullae or blebs on the pleural surface, or any pathology which may result in adhesions of the pleural and parietal pleura are examples of some other clinical conditions. Note that we are quite able to rule out some of these conditions, but for the others, there is no ready means to identify them.

Determination of the effects of a decompression on pulmonary tissue is fraught with difficulty. Hypoxia, low pressure effects, disease, biologic variations and poor study design have limited the usefulness of data collected in this area. They can affect future studies if steps are not taken during study design to eliminate or control for their effects.

With these limitations in mind, the last section discusses some specifics of the human and animal data.

DISCUSSION

High altitude flight in advanced fighter aircraft without pressure suits will require both increases in oxygen content and pressure. Alveolar oxygen content may be maintained through increase in pressure, increase in oxygen content, or both.[12] At 37,500 feet, 100% oxygen without additional pressure will maintain an oxygen pressure of 159 mmHg.[77,122] Above this point, additional pressurization will be required in the form of either cabin pressurization, or directly applied positive breathing gas pressure, or both.[50] Both methods have implications for pulmonary over-pressure.

The physiologist and engineer control barometric pressure change around several parameters.[48,77] Primary human factors relating to the choice of cabin altitude are Decompression Sickness (DCS), hypoxia and explosive decompression. DCS is a primary factor determining maximum cabin altitude without a pressure suit. In general, to prevent DCS cabin altitude should not exceed 22-24000 feet (321-294 mmHg) for flights of short duration.[55] Another factor contributing to the

decision of maximum allowable cabin altitude is explosive decompression. The greater the differential between the cabin altitude and the ambient aircraft altitude, the greater the potential for injury to the pilot from decompression.[48] All decompressions to altitudes in excess of 40,000 feet (141 mmHg) may be considered as decompressions terminating at this pressure altitude if the human is to survive. The prevention of hypoxia in the steady state at these heights demands that the absolute pressure within the respiratory tract not fall below the equivalent of 40,000 ft, 141 mmHg.[77,122] What must be considered in decompression to altitudes above 40,000 feet (141 mmHg) is the peak pressure experienced by the pulmonary system, and the life support equipment designed to ensure adequate oxygen pressure and chest counter-pressure.

In addition to use of pressure to maintain adequate alveolar oxygen at altitude, positive pressure breathing for G protection is now being employed in our combat forces.[15,62,64,71,88,92,108] While pressure is theoretically limited to safe static pressure, the possibility of exposure to over-pressure exist. This may occur through dynamic overshoot of the system, or through cabin pressurization failure during high G maneuver. Knowledge of a safe dynamic over-pressure would aid designers in providing a maximally effective system for lowest cost.

Concepts of decompression theory, physiology, research problems, and currently used guidelines have already been explored. This section reviews the animal and human studies which form the limits of the experimental data. First animal and human limits will be explored. Briefly, animal studies which explore the realm of multiple decompressions are reported. Finally, studies which explore human tolerance to decompression, with flight equipment, are presented.

Can the peak pressure limit be quantified? Damon et al. used 20 dogs to assess the effects of intermittent positive pressure breathing (IPPB) after exposure to blast in a controlled experiment.[41] Peak pressure was in the range of 2590 mmHg in about 0.3 seconds. Mortality was greater than 50% in this group. Because of the extreme nature of the exposure, this experiment is not readily comparable to other data. This study did demonstrate that there is a limit of exposure which can be quantified in terms of mortality. The problem for researchers to address, is a clear definition of the desired limit. What is an acceptable risk to our pilots? In this study, the researchers were looking for a 100% lethal decompression. Due to technological limitations, they were not able to reach this, however a 50% lethal decompression was reached.

Hall got closer to an animal limit.[67] This was the experiment which set the animal limit on Graphs 5 and 6. The experiments tested the theory that some finite time is required for the elastic tissues to elongate and cause damage. The author attempted to find the rate and magnitude of explosive decompression required to produce lethal effects in albino rats. This was a well designed experiment without major design flaws. All three control groups of five animals each remained above 18,000 feet (379 mmHg) for 5 seconds or less. In the explosive decompression groups, time above 18,000 feet (379 mmHg) was limited to 2 seconds, and return time to sea level was less than 26 seconds in all groups. In the first control group, sea level (760 mmHg) to 40,000 feet (141 mmHg) in 0.53 second, all animals remained normal. In the second control group, sea level (760 mmHg) to 69,000 feet (35 mmHg) in 0.9 second, the animals developed slight

pulmonary ecchymosis, but no hemorrhage. The ears were mildly affected. The third control group, sea level (760 mmHg) to 105,000 feet (7 mmHg) in 1.1 seconds, was the same with respect to the lung, but had more ear effects, with none to moderate dilation of the auricle. In the experimental groups, auricular damage was minimal in all groups. Death rate in the first experimental group, sea level (760 mmHg) to 40,000 feet (141 mmHg) in 0.004 second, was 40%. Pulmonary damage was mild to moderate hemorrhage. Some epistaxis was present. Death rate in the second experimental group, sea level (760 mmHg) to 69,000 feet (35 mmHg) in 0.006 second, was 70%. Pulmonary damage was moderate to massive hemorrhage. Massive epistaxis in 70% as well. In the last group, sea level (760 mmHg) to 105,000 feet (7 mmHg) in 0.007 second, mortality was 70%. Pulmonary hemorrhage was moderate to massive.

The authors conclude from the control groups that the process of explosive decompression does produce some pathological change in the absence of anoxia and decompression sickness. However, the authors realized from the experimental groups that a patent tracheal passage might allow for dissipation of pressure in the lungs no matter how rapid the onset of pressure change. If there was a minimal finite time to allow for expansion of alveolar tissue in the rat, it must be less than 0.004 seconds for 100% mortality.

Practically, the study demonstrated no mortality with decompression from sea level (760 mmHg) to 105,000 feet (7 mmHg) in about one second. Seventy percent mortality was experienced when the same decompression range was experienced in about 0.01 second. This narrows the limit for the rat for a one atmosphere decompression to between 0.01 and 1 second, for complete survival.

Two animal experiments demonstrated that decompressions of a severe nature could be tolerated, Vail, 1952, and Gelfan, 1951.[61,113] Vail explored the forces in the thorax during explosive decompression.[113] The experiment used eight dogs under nembutal anesthesia. They were exposed to a 10 psi decompression, 10,000 feet (523 mmHg) to 72,000 feet (30 mmHg) in 0.15 second. Repressurization was accomplished within 30 seconds to minimize the effects of hypoxia. Each animal was subjected to three decompressions with 30 to 45 minutes between decompressions. No deaths occurred as a result of the decompressions. The chest was unsupported. All animals survived. The highest pressures were recorded on the initial decompression. The average intrapleural pressure increase was 95.7 mmHg, the maximum, 135 mmHg, the minimum, 68 mmHg. No pneumothorax was observed at autopsy. Average tracheal pressure was 53 mmHg, range 28-100 mmHg. All animals autopsied after explosive decompression were found to contain isolated petechial hemorrhage, and areas of atelectasis. Animals allowed several days to recover after the explosive decompression did not show any evidence of hemorrhage or atelectasis. Vail concluded that the pressures encountered in this study are capable of causing pulmonary pathology.

Gelfan's primary objective was to determine survival rates from exposure to an explosive decompression and free fall descent from extreme altitude.[61] 105 monkeys were exposed to a total of 202 decompressions, sixty-four percent were to over 70,000 feet (34 mmHg). He demonstrated however, that animals can survive explosive decompression to simulated altitudes

of as high as 80,000 feet (21 mmHg) if recompression is rapid enough. In general, explosive decompression from 20,000 feet (335 mmHg) to 77,000 feet (24 mmHg) is survivable.

Determination of human decompression limits is confounded by the same issues of hypoxia, ebullism and decompression sickness. However, additional issues must be considered.[27,54] Some of the difficulties related to research at 40,000 to 50,000 feet (141 to 87 mmHg) were stated in 1961, and remain true today.[27] Exposure to altitude above 40,000 feet (141 mmHg) is hazardous, even in an experimental situation. Valid statistical data on a large number of subjects is difficult to obtain due to the safety requirements which usually necessitate single subject runs. In order to identify the limit of safety, some toxicity will occur. The long term effects, if any, of the toxicity are not known.

Early work in the field presented only a single observation.[13,38,40,60,72] In an argument for the safety and necessity for development of pressurized cabins, the author states that laboratory personnel "have been brought to a 15,000 feet altitude in 0.01 second without harmful after effects." In an introduction to animal experiments, which were well out of the range of our interest, the authors state that they could tolerate the 620 mmHg decompression in 4 seconds without discomfort or ill effects. This is only a single observation, no medical examination is documented.[60]

Three papers described human tolerance to decompression without flight gear. Sweeney's experiments are noteworthy for the severity of the decompressions used.[54,110] One hundred fifty decompressions were conducted from 9,800 feet (527 mmHg) to 35,000 feet (179 mmHg) in 0.1 second. Fifteen experiments exposed human subjects to a decompression from 8,000 feet to 35,000 feet (564 to 179 mmHg) in 0.1 second. Ten experiments decompressed subjects from 27,000 to 45,000 feet (258 to 111 mmHg) in 0.015 second. No ill effects were noted. As I have been unable to obtain the original paper, it is not clear the extent to which ill effects were ascertained.

Hitchcock et al., 1948, designed experiments to directly test the tolerance of normal subjects to explosive decompression.[79] In addition, they determined the effect of explosive decompression on the incidence and susceptibility to decompression sickness. 150 human subjects were subjected to both rapid and explosive decompression. Subjects ranged from 16 to 56 years of age, with the majority between 18 and 25 years. In all, 500 experiments with rates as fast as 1300 mmHg per second over ranges of half an atmosphere caused no apparent ill effects. The most frequent EKG finding was sinus tachycardia. X-rays pre and post decompression were compared on all subjects. No pulmonary lesions were noted. Subjects were able to carry on required activities following the decompression during the 15 to 90 minute period at low pressure. The highest altitude of exposure was 45,000 feet (111 mmHg). The highest decompression rate was 1320 mmHg per second. The authors concluded that decompression within the rates and ranges experienced during the study does not constitute a serious hazard to normal human beings.

Multiple decompressions were found to be more hazardous than single decompression of the same magnitude. Edelmann et al., 1946, in an uncontrolled, and poorly designed study, sought to isolate the effects of explosive decompression from the effects of hypoxia.[45] All dogs survived the multiple decompressions, however, pulmonary hemorrhage was more pronounced in the group multiply exposed. Burkhardt et al. used dogs to explore the effects of explosive decompression.[31] The 26 dogs decompressed once through a "safe" range, 338 mmHg in 0.125 second, did experience some pulmonary hemorrhage by pathology exam compared with dogs that were not decompressed. This provided good experimental evidence that subclinical damage can occur, and may not be evident on gross exam. In order to explore the effects of multiple "safe" decompressions, 81 dogs were decompressed through this range seven times in several days. The 81 dogs experienced significantly more hemorrhage than the control group and the group of 26 dogs exposed to one decompression. In further experiments, dogs exposed to multiple decompressions while wearing pressure breathing masks had even greater amounts of pulmonary hemorrhage.

Repeated exposures to a "safe" explosive decompression may be more severe than a single exposure to RD.[31,45,121] Most of the experimental evidence in this review explores the effects of single decompression. There is sufficient evidence that decompressions occurring sufficiently close in time may result in additive damage. The questions of whether the healing process will result in complete resolution of the pulmonary damage sustained, or the minimal interval between rapid or explosive decompressions remain unanswered. There are no human studies on the pulmonary toxicity of multiple "safe" decompressions to validate these results.

In addition to rate and range of pressure change, it is also important to consider the effects of life support equipment.[54,85] Animal and human studies have been performed. Rosenbaum, 1957, reported explosive decompression studies with animals wearing full bladder pressure vest and helmet.[100] Decompressions were from 8,000 feet (564 mmHg) to 65,000 feet (43 mmHg) in 0.03 second. There are no other published studies of animals wearing chest and abdominal binding and breathing a safety pressure from an automatic regulator. 17 animals were available--dogs. Appropriate controls were used, and thorough health screening of the subjects was performed.

Dogs were chosen between 10-14 kg. in size, held in quarantine for 28 days and given a full veterinary evaluation. They were then fitted repeatedly with the mask and pressure vest, as well as exposed to routine chamber noises to acquaint the animals to the testing environment. All decompressions took place wearing a full bladder suit and breathing helmet. The mask and chest counter-pressure were intimately connected by a compensated breathing valve, which kept chest pressure and mask pressure within 15 mmHg of each other throughout the decompression. All animals survived. Some animals were sacrificed for autopsy exam, and no gross lesions were found. Other animals were considered healthy by serial physical exams, blood studies and CXR for 6 weeks following the decompression. In the decompressions from 8,000 feet to 65,000 feet (564 to 43 mmHg) in 0.028 second, pressure in the bladder and helmet were measured. Pressures peaked at 235 mmHg, and were never more than 15 mmHg apart. There was thus never greater than 15 mmHg pressure in the lungs. These animals did not have any evident gross pathology.

Four control animals decompressed without altitude protection gear and autopsied showed considerable atelectasis and apical emphysema.

A pressure tracing from the 1957 Rosenbaum report is included here as figure 2. It is the best experimental evidence that suggests the possibility of a pressure vest system which can support the decompression of a human through a 14 and 10 psi (720 and 520 mmHg) pressure. Further experimentation in this area would be key in exploring a 7 psi (363 mmHg) differential cockpit to reduce the incidence of DCS at altitude.

The British, in 1942 outlined some operational requirements for flight gear.[7] Their experiments in explosive decompression were performed to determine the effects upon personnel exposed. Maximal operational altitude at the time was 45,000 feet (111 mmHg). Cabin pressure differentials were 7 psi (363 mmHg), but were reduced to 2.5 psi (130 mmHg) where enemy action may be encountered. The authors chose 5 psi (259 mmHg) differentials for the experiments, and 45,000 feet (111 mmHg) as the max altitude, as these represented the operational extremes expected to be encountered.

Thirteen decompressions were performed on four individuals. There were several complaints of localized chest wall pain and attacks of coughing, but no serious pulmonary damage. The authors report on the gastrointestinal tract, the ears and sinuses, as well as the cardiovascular system. No abnormalities are reported. The maximum pressure change was 5.8 psi (300 mmHg), with little or no subjective discomfort. The authors conclude that a pressure ratio of 2.5:1 is the maximum to be allowed for experimental purposes, but that a greater ratio could probably be tolerated without gross physical changes.

Burkhardt et. al. collected human data to demonstrate that pressures within masks worn during explosive decompressions must be considered.[31] The human exposures were to known "safe" decompression ranges of 3 and 5 psi (156 and 262 mmHg). There were no ill effects by subject report.

Ernsting et al., in 1960, assessed the risk of decompression from 20,000 feet to 50,000 feet (349 to 87 mmHg) in 0.1 second to a person wearing flight equipment.[52] This change in pressure is the 5 psi (262 mmHg) now assumed to be a safe exposure. The study performed was well controlled, and well designed. The investigators thoroughly investigated the performance of each piece of the flight equipment separately and in combination using a simulated lung prior to the human experiments. There were some significant differences in measured parameters between the simulated and human experiments. In most instances the pressure in the simulated lung was higher than the human experiments due to the rigidity of the simulated lung.

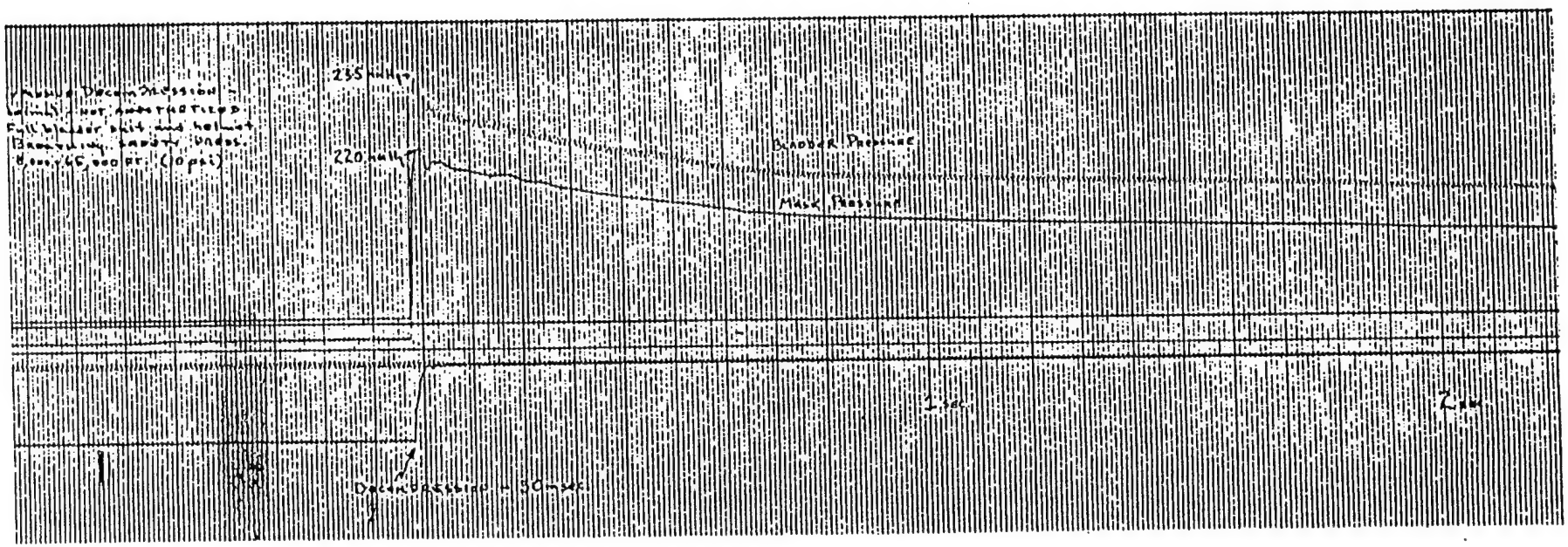


Figure 2. EXPLOSIVE DECOMPRESSION - ANIMAL NOT ANESTHETIZED - WEARING FULL BLADDER SUIT AND HELMET - BREATHING SAFETY PRESSURE - 8,000 to 65,000 feet (10 psi)

While Ernsting's experiments were not designed to test the limits of human tolerance, they did suggest an experimental approach which could be used to establish the limit of tolerance. It is important to note that one of the principle advantages is the incorporation of the equipment to be used in the flight environment within the experimental design. For example, the pressure jerkin was supposed to provide chest counter-pressure during the decompression. However, in the course of the experiments it was realized that the particular design of the pressure jerkin allowed no significant support to the chest wall during the decompression due to inflation characteristics. This jerkin provided no protection to over-distension of the lungs. These experiments were successful in providing the basis for an experimental methodology for exploring the limits of human tolerance.

Luft et. al., 1953, measured pressure imposed upon the lungs and chest during explosive decompression at high altitude while wearing pressure-demand oxygen equipment.[86] The authors recognized the potential for peak pulmonary pressure in excess of human tolerance if the mask does not provide adequate pressure relief. The experiments were carefully controlled and well planned.

A total of 35 decompressions were performed on five subjects. The range of pressures experienced were from 20-25,000 feet (349 to 282 mmHg) to 47-50,000 feet (101 to 87 mmHg) over 0.3 to 0.5 seconds during normal breathing while wearing a pressure-demand oxygen mask. Mask pressure ranged 38-84 mmHg, with an average of 61 mmHg. Pressure relief was obtained through leakage around the mask. This was confirmed in a test to 61,500 feet (50 mmHg) which did not yield pressures significantly different from the other experiments. The peak mask pressure for this experiment was 72 mmHg while the peak intrathoracic pressure was 74 mmHg. No subjective or objective evidence of pulmonary damage was found during or after the experiments. The authors concluded that the pressure breathing masks type A-13, A-14 with the D-1 regulator did not pose a hazard to aviators in the explosive decompression environment within that expected in the flight envelope they would be employed.

The primary objective of Balldin's 1976 study was to evaluate flight equipment during explosive decompression from a medical safety point of view.[16] The study primarily looked for extra-pulmonary gas leakage and the appearance of bubbles within the vascular system. The subjects were wearing a two piece flight suit which consisted of a chest counter-pressure garment and an anti-G suit which was pressurized to a pressure 3.2 times the breathing pressure. The mask pressure was balanced by an inflatable bladder in the rear of the helmet. Peak pressure was 70 mmHg at 65,600 feet (41 mmHg). Seven human subjects were exposed to a total of ten explosive decompressions. After one hour of oxygen breathing, the subjects were explosively decompressed from 29,500 feet (231 mmHg) to 57,400 feet (61 mmHg) or 65,600 feet (41 mmHg) in 0.5 sec. Subjects stayed at altitude for only 30 seconds. No DCS or gas embolism was seen. Chest x-ray at sea level after the exposure did not reveal any abnormality. Although this study did not locate any pulmonary air leaks, they cite the problems: any gas leaked would be compressed on the return to sea level; the breathing gas was oxygen and would be absorbed quickly. This study would have benefitted from an x-ray at altitude.

The altitudes chosen were at the maximum differential believed to be safe, 190 mmHg. Peak pulmonary pressure never rose above ambient plus 70 mmHg added by the system. There was a small delay in the onset of positive pressure to the peak of 70 mmHg. These were safe decompressions, and did not approach the limits of human tolerance.

It should be noted that all of the data presented indicate completed decompressions without permanent injury, except where noted. The range of decompressions was from 0.01 second to over 5 seconds, with pressure changes of only 100 mmHg to almost one full atmosphere. In general, humans were not exposed to conditions beyond what might be expected in operational conditions.[85] This points the way for future research requirements. To date, the exact limits for safety for explosive decompression have not been defined. The boundaries of the human experience are evident on all graphs.

CONCLUSIONS

The papers reviewed here define the currently understood limits for human pulmonary over-pressure. Animal models were used to validate theory and physiologic pulmonary events of decompression. The animal data provides evidence for a decompression limit; evidence that extreme conditions may be survivable; evidence that damage attributed to decompression at altitude is often due to anoxia, and suggests repeated decompressions are more hazardous than a single decompression. In addition, animal data confirms flight equipment can be either protective or hazardous to the subject of a decompression. Controlled, experimental data on human tolerance to dynamic pulmonary over-pressure is limited to a few studies. These studies do not suggest that the human tolerance for dynamic pressures is significantly different from the animal models used. Carefully constructed studies will be required to define the limit of pulmonary toxicity in the dynamic over-pressure scenario. Determination of this limit is vital to maximize our performance capabilities in operational aircraft.

The following research questions need to be addressed to begin to define the limit:

1. What is the shape of the curve defining the biologic variability surrounding pulmonary pressure limits?
2. What is the pressure that we expect out aircrews to be exposed?
3. What is the incidence of pulmonary and pleural pathology in the aircrew population?
4. What is the best screening tool for aircrew selection regarding occult pulmonary pathology?
5. What is the performance of current life support equipment in the explosive decompression environment at high altitude?

6. Currently no model is available to adequately predict the outcome of a specific decompression except to plot it on a graph of currently explored environments.

Policy questions which need to be addressed:

1. What is the fatality rate from pulmonary over-pressure we are willing to accept?
2. What are the mission requirements for the aircraft?
3. Are we willing to consult the life support and human physiology divisions prior to setting aircraft life support specifications?

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APPENDIX A

Appendix A: Experiments in Explosive Decompression, Animal Subjects

Number of Experiments	RGE	Pc/Pa	Pc-Pa	Pc (mmHg)	Pa (mmHg)	Time (S)	Remarks
Bancroft and Dunn, 1965, Dogs							
18	25	90	178	180	2	.2	O_2 Atmosphere
8	25	90	178	180	2	1	
Burkhardt, Coulson, Criscuolo, Adler, 1951, Dogs.							
26			338.7			.125	
81			338.7			.125	
Damon, Henderson, Jones, 1973, Dogs.							
20			2587.5			.140	
Edelmann, Whitehorn, Lein, Hitchcock, 1946, Dogs.*							
1	11.90	6.01	436.00	523	87	.012	$MgSO_4$
2	5.06	3.71	382.00	523	141	.011	Electrocute
8	11.90	6.01	436.00	523	87	.012	Electrocute
10	11.90	6.01	436.00	523	87	.012	Nembutal
4	11.90	6.01	436.00	523	87	.09	Nembutal
2	11.90	6.01	436.00	523	87	.36	Nembutal
1	11.90	6.01	436.00	523	87	.03	Nembutal

* Remarks refer to mode of sacrifice.

Appendix A, Continued: Experiments in Explosive Decompression, Animal Subjects

Experiments	RGE	Pc/Pa	Pc-Pa	Pc (mmHg)	Pa (mmHg)	Time (S)	Remarks
Rhesus Monkeys.							
36	25	13.85	323.90	349.1	25.2	.2	
94	25	11.48	318.70	349.1	30.4	.2	
22	25	9.07	310.60	349.1	38.5	.2	
16	18.9	5.54	286.10	349.1	63	.2	
7	14.6	6.57	443.00	522.6	79.6	.2	
1	13.2	7.52	659.00	760	101	.2	
17	8.0	5.59	624.00	760	136	.2	
Edelmann and Hitchcock, 1952. Dogs.							
40	25	25.00	720.0	750	30	.03	15,30,45,60 Seconds at altitude.
10	25	25.00	720.0	750	30	.03	2 Minutes, 4 Deaths
2	25	25.00	720.0	750	30	.03	4 Minutes, 2 Deaths
Grognot and Senelar, 1958. Dogs.							
		3.1				.03	
Fegler, FPRC 349, 1941, Guinia Pigs.*							
54	8.4	5.73	619.0	750	131	.5	8 Deaths, No Oxygen used.
8	8.4	5.73	619.0	750	131	.5	0 Dead, 2 Prebreathed Oxygen
10	11.2	6.82	640.0	750	110	.5	5 Dead, No Oxygen used.
16	11.2	6.82	640.0	750	110	.5	0 Dead, 30 Minutes Oxygen
16	4.5	3.84	710.0	960	250	.5	0 Dead

* Remarks refer to number of animals dead, use of oxygen during recovery. and to use of oxygen prebreathing period.

Appendix A, Continued: Experiments in Explosive Decompression, Animal Subjects

Number of Experiments	RGE	Pc/Pa	Pc-Pa	Pc (mmHg)	Pa (mmHg)	Time (S)	Remarks
Hall, 1957, Rats.							
5	7.61	5.40	619.30	760	140.7	0.53	Normal
5	21.3	21.71	725.00	760	35	0.9	Slight Damage
5	108.6	108.57	753.00	760	7	1.1	Slight Damage
10	7.61	5.40	619.30	760	140.7	.0043	4 Dead, Moderate Damage
10	21.3	21.71	725.00	760	35	.0068	7 Dead, Moderate to Severe Damage
10	108.6	108.57	753.00	760	7	.0075	7 D, Moderate to Severe Damage
Hall and Corey, 1950. Albino Rats.*							
20	25	36.19	739.00	760	21	.64	1 second
12	25	36.19	739.00	760	21	.64	40 seconds
10	25	36.19	739.00	760	21	.64	40 seconds, 8 Dead
Rosenbaum, 1957, Dogs.							
8	25	13.25	521.80	564.4	42.6	.028	Helmet
6	25	22.62	726.40	760	33.6	.030	Helmet
3	25	13.25	521.80	564.4	42.6	.028	Helmet
4	25	13.25	521.80	564.4	42.6	.028	Control
Vail, 1952, Dogs.							
8				523	30	.15	

Remarks refer to time at altitude.

APPENDIX B

Appendix B: Experiments on Explosive Decompression, Human Subjects

Number of Experiments	RGE	Pc-Pa	Pc (mmHg)	Pa (mmHg)	Time (S)	Remarks
Sweeney, 1944						
2	2.33	53.4	140.7	87.3	0.011	
3	2.36	66.4	162.4	96	0.012	
9	2.20	76.4	187.3	110.9	0.012	
10	3.31	147.1	258	110.9	0.015	Approaching Tolerance Limit
150	3.64	347.9	526.6	178.7	0.101	
15	3.93	385.7	564.4	178.7	0.106	
Garsaux, Richlou, Laurent, 1939						
	7.62	619.3	760	140.7	4.0	
Heim, 1938						
	1.86	331.2	760	428.8	0.01	
Balldin, 1978						
5	13.19	169.6	230.6	61	.5	Mask, G-Suit, PPB
5	Undefined	189.6	230.6	41	.5	
Ernsting, Roxborough, Wagner, 1960						
1	1.93	138.3	334.6	196.3	0.07	Pressure Helmet, G-Suit, and Jerkin on Subjects
1	1.93	138.3	334.6	196.3	0.08	
7	1.93	138.3	334.6	196.3	0.09	
3	2.33	164.3	334.6	170.3	0.10	
2	2.67	179.8	334.6	154.8	0.11	
1	4.51	223.7	334.6	110.9	0.17	Peak Helmet Pressure 38 mmHg

Appendix B, Continued: Experiments in Explosive Decompression, Human Subjects

Number of Experiments	RGE	Pc-Pa	Pc (mmHg)	Pa (mmHg)	Time (S)	Remarks
Clamann, Luft, Adler, 1948						
3	2.00	354.8	760	403.2	2	Mask, Nose Plugged, Room Air
2	2.33	406.50	760	353.50	2	
3	2.57	435.10	760	324.9	2	
3	2.74	452.60	760	307.4	2	
2	3.35	499.80	760	260.2	2	
1	3.88	529.40	760	230.6	2	
4	4.29	546.60	760	213.4	2	
2	4.72	561.80	760	198.2	2	
6	4.78	563.70	760	196.3	2	
5	5.25	577.00	760	183.0	2	
6	7.35	615.90	760	144.1	2	
1	7.62	619.30	760	140.7	2	
9	12.08	653.90	760	106.1	2	
8	12.18	654.40	760	105.6	2	
3	18.71	674.80	760	85.2	2	
5	4.17	476.40	673.6	197.2	2	Mask, Nose Plugged, On Oxygen
6	6.41	528.80	673.6	144.8	2	
3	8.79	555.20	673.6	118.4	2	
3	9.28	559.00	673.6	114.6	2	
2	12.35	575.80	673.6	97.8	2	
2	15.58	586.30	673.6	87.3	2	
1	3.71	109.00	196.3	87.3	2	
2	4.92	118.90	196.3	77.4	2	

Appendix B, Continued: Experiments in Explosive Decompression, Human Subjects

Number of Experiments	RGE	Pc-Pa	Pc (mmHg)	Pa (mmHg)	Time (S)	Remarks
Burkhardt, Colsen, Crisculo, Adler, 1951						
1		121			0.44	A-15 Mask
1		143			0.59	A-15
1		153			0.5	A-15
1		238.4			0.84	A-15
1		241			0.84	A-15
1		237			0.81	A-13
1		236			0.69	A-13
1		299			1.0	A-13
Billings, Ernsting, 1974						
24	3.93	385.3	564.4	178.7	2	
24	4.49	402	564.4	162.4	2	
24	5.15	416.9	564.4	147.5	2	
24	5.95	430.3	564.4	134.1	2	
F.P.R.C. 437, 1942						
3	1.12	77.3	751.8	674.5	~.8	
3	1.18	106.00	751.8	645.8	~.8	
3	1.24	135.80	751.8	616.0	~.8	
1	1.60	139.60	420.2	280.6	~.8	
1	1.85	138.30	347.7	209.4	~.8	
1	1.58	140.80	428.8	288.0	~.8	
1	1.83	110.20	290.6	180.4	~.8	
1	2.62	143.00	278.2	135.2	~.8	
1	2.54	201.60	379.4	177.8	~.8	
1	2.80	194.30	349.1	154.8	~.8	
1	3.40	165.60	281.8	116.2	~.8	
1	2.16	244.60	502.6	258.0	~.8	
1	2.74	302.20	522.6	220.4	~.8	

Appendix B, Continued: Experiments in Explosive Decompression, Human Subjects

Number of Experiments	RGE	Pc-Pa	Pc (mmHg)	Pa (mmHg)	Time (S)	Remarks
Hitchcock, Whitehorn, Edelmann, 1948						
13	3.92	385	564	179	2.2	
13	3.92	385	564	179	1.3	
15	3.92	385	564	179	.6	
30	3.92	385	564	179	.4	
46	2.22	262	523	261	1.5	
19	2.22	262	523	261	.2	1310 mmHg/s
36	3.61	344	523	179	8.0	
90	3.61	344	523	179	2.0	
4	3.61	344	523	179	0.3	1147 mmHg/s
2	5.07	382	523	141	8.9	
3	5.07	382	523	141	2.2	
22	2.29	170	349	179	4.5	
3	2.29	170	349	179	1.1	
66	3.21	208	349	141	5.5	
91	3.21	208	349	141	1.4	
8	3.21	208	349	141	0.7	
7	3.21	208	349	141	0.3	
20	3.21	208	349	141	0.2	1040 mmHg/s
66	3.22	142	253	111	2.0	

Appendix B, Continued: Experiments in Explosive Decompression, Human Subjects

Number of Experiments	RGE	Pc-Pa	Pc (mmHg)	Pa (mmHg)	Time (S)	Remarks
Luft, Bancroft and Carter, 1953.						
1	5.20	243.00	348	105	.4	(.3-.5) A-14 Regulator A-13 Mask Pressure Breathing
1	5.67	247.00	347	100	.4	
1	7.60	270.00	358	88	.4	
1	6.25	257.00	353	96	.4	
1	7.33	259.00	347	88	.4	
2	4.44	182.00	282	100	.4	
1	4.49	181.00	280	99	.4	
5	4.78	200.00	300	100	.4	
5	4.78	200.00	300	100	.4	D-1 Regulator, A-13 Mask (.3-.5s)PPB
7	6.34	213.00	300	87	.4	
3	6.51	220.00	307	87	.4	
1	6.29	211.00	298	87	.4	
1	6.26	210.00	297	87	.4	
1	6.31	212.00	299	87	.4	
1	4.71	159.00	249	90	.4	
1	6.50	214.00	300	86	.4	
1	7.90	227.00	307	80	.4	

RGE=(Pc-47.09)/(Pa-47.09)